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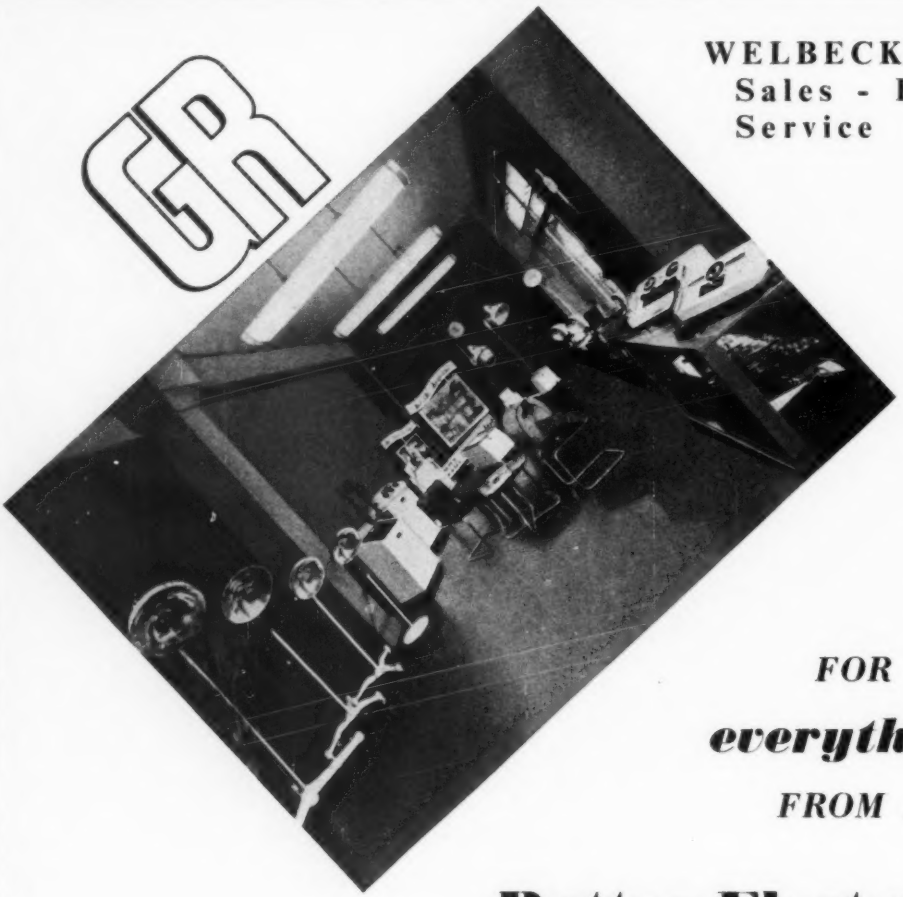

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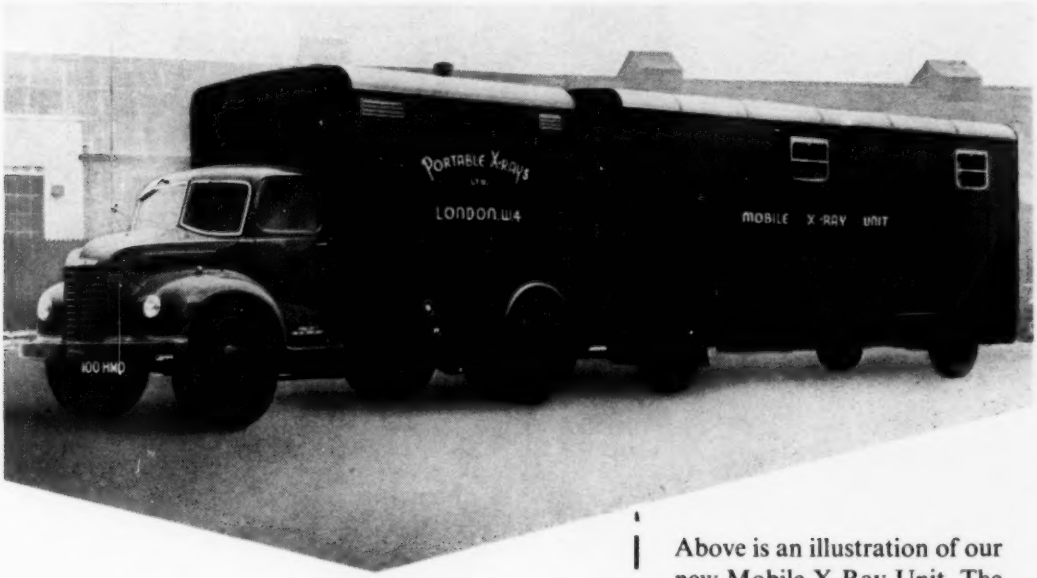

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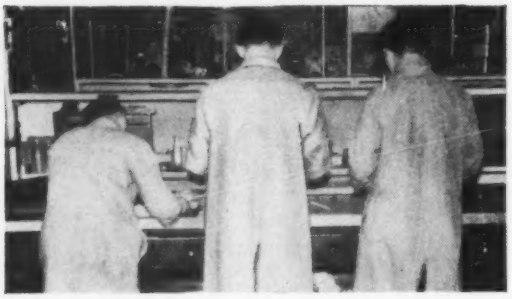

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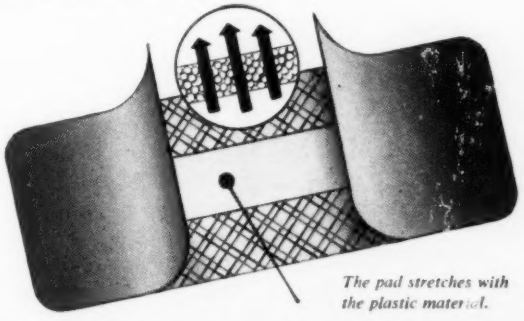

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Brit. J. industr. Med., 1957, **14**, 1

THE DANGER OF DERMATOSES DUE TO COLD-SETTING ETHOXYLINE RESINS (EPOXIDE RESINS)

BY

E. GRANDJEAN

From the Institute of Hygiene and Industrial Physiology of the Swiss Federal School of Technology, Zurich

(RECEIVED FOR PUBLICATION JULY 24, 1956)

A new group of synthetic resins (the ethoxyline or epoxide resins) has been widely accepted in industry during the last few years. These resins, thanks to their novel properties as adhesives and casting resins, are used in the modern manufacture of various kinds of apparatus, of electrical equipment, of automobiles, and of aircraft. They are usually supplied in the form of two reactive components, resin and hardener.

The cold-setting casting resins occupy a special position among the ethoxyline resins, most of which are aliphatic-aromatic polyethers containing hydroxyl and terminal epoxy groups. The highly reactive hardeners employed are usually polyamines. Mixtures of these two are capable of transformation at room temperature into solid, infusible substances. In practice, the ethoxyline resin is mixed with the polyamine hardener in the ratio of about 10 : 1 immediately before use and then applied to the desired point of the material by casting or brushing or injecting.

The ethoxyline resins were introduced under the general term " epoxide or epoxy " resins, being offered under the trademarks "epon" or "araldite" casting resin. As is well known, both manufacturers and users of lacquers are familiar with the handling of dangerous substances and apply comprehensive protective measures; as a result few or no dermatoses have appeared in lacquer factories till now. Dermatoses were reported in the U.S.A. too after these products had been found useful in various industries as adhesives and casting resins.

The introduction of ethoxyline resins into works hitherto unfamiliar with chemical processes brought the operatives into contact for the first time with substances which carried an unknown risk to the skin. Therefore, the first extensive " epidemics " of skin eruptions appeared mainly in such " non-chemical " factories.

The first observations of skin irritation during work with such resins were reported by Pletscher, Schuppli, and Reipert (1954). Six out of 20 operatives working with " araldite " casting resin D suffered from severe eczema, eight exhibited only slight, temporary epidermal irritation, and the rest remained free of symptoms. Since the epidermal symptoms were apparently of an allergic nature, the 20 operatives were patch-tested for epidermal sensitivity to " araldite " casting resin D, hardener 951 (triethylenetetramine), and various other substances. These tests showed that out of the 14 workmen suffering from epidermal reactions, four were hypersensitive to " araldite " casting resin D, two to hardener 951, and three to both substances. The men free from symptoms exhibited no hypersensitivity. On the basis of their observations, the authors concluded that contact with " araldite " casting resin D and hardener 951 can lead to allergic epidermal eczema. The present paper describes our investigations in 11 factories in four European countries (mainly in electrical engineering) using these substances.

Atmospheric Pollution by Vapours of Triethylenetetramine

Araldite resins are practically non-volatile and are therefore only present in traces in the surrounding atmosphere. We therefore determined the concentration of triethylenetetramine (T.E.T. = hardener 951) in the atmosphere as a measure of the degree of pollution of the air at the work bench.

Method.—The air to be analysed was passed through two absorption tubes filled with glass beads. For each analysis, portions of up to 160 litres of air were passed over a period of several hours. We employed either 0·1 NHCl or cation- and CO_2-free distilled water as the absorbing agent. The absorbed T.E.T. was then determined quantitatively in the laboratory with the amine test reaction using 1 : 2-naphthoquinone-4-sulphonic acid according to the method of Englis and Fiess (1944).

Experiments.—In a first series of experiments, tin lids were filled at short intervals during eight hours with a mixture of araldite casting resin D/T.E.T. in an unventi-

lated room of 16·5 c.m. volume. On a model work bench, the quantities of araldite casting resin D thus cast per day amounted to 1,800 to 4,200 g. and the total surface cast per day to about 5,000 to 15,000 cm². This exceeded the amount usually employed per day on one factory work bench.

The T.E.T. concentration measured at head level at our model work bench varied between 0·31 and 0·59 μg./l. air. We tried in a similar manner to measure the T.E.T. concentration at head level above two work benches in a condenser factory in which condensers were continuously cast. The total surface of " araldite " casting resin D cast daily varied between 1,500 and 3,500 cm², and the quantities processed daily per operative between 3,400 and 3,600 g. In spite of the fact that more than 100 litres of air were drawn through the absorption tubes for each analysis, the T.E.T. concentrations measured never exceeded the lower limit of analysis of about 0·15 μg./l. air. We concluded that at a normal work bench the average concentration of T.E.T. vapour at head level is below the limit of measurement of 0·15 μg./l. air.

Working Conditions at Work Benches in 11 Factories Studied

The nature of the operations in these factories was as shown in Table 1. Whereas the ratio of resin to hardener was in all factories 10 to 1 (in some cases quartz, slate, or porcelain was added to the resin), the manner of mixing varied from automatic methods and vibromixers to manual mixing with spatulae.

TABLE 1

THE APPLICATIONS OF ARALDITE CASTING RESIN D IN 11 FACTORIES INVESTIGATED

No. and Description of Factory	Particular Use of Araldite Casting Resin D
1 (condenser factory)	Sealing condensers
2 (condenser factory)	Sealing condensers
3 (electrotechnical factory)	Sealing condensers Coil assembly
4 (electrical apparatus works)	Sealing condensers Casting switch boxes and contact connexions
5 (manufacture of telephones and other electrical apparatus)	Sealing condensers Casting coils and lead couplings Coil assembly
6 (telephone factory)	Sealing condensers
7 (electrical apparatus works)	Sealing condensers Coil assembly
8 (refrigerator factory)	Refrigerator assembly
9 (manufacture of telephones and other electrical apparatus)	Sealing condensers
10 (cable factory)	Sealing cable ends
11 (manufacture of light bulbs and other electrical appliances)	Sealing condensers

The modes of applying the resin vary widely, *e.g.*, manual casting (for large condensers), mechanical casting (for small condensers), manual application (in assembly of coils in switch boards) with immediate removal of exuded excess or later tooled removal after setting, pouring of resin on to parts of a frame to be bonded (refrigerator assembly) with later removal of excess. Some factories had installed ventilator hoods over casting work benches.

On inspecting the techniques employed, the work benches were always found to be contaminated as well as tools, clothes, and the hands of the operatives. The containers for the resin are often very unsatisfactory and permit it to overflow on to the bench during pouring. The women wipe up the mess with their fingers. Stirrers, spatulae, brushes, and other implements with much adherent resin are laid on the benches. These and other causes increase the probability of skin contamination, which is further increased by the use of cleansing solvents (acetone, alcohol, butanone, trichloroethylene, and mixtures of aromatic hydrocarbons).

Clinical Aspects of Epoxy-resin Dermatosis

These have been variously described. Plüss (1954) and Pletscher *et al.* (1954) classify them as industrial eczemas, usually on the hands and forearms, with oedema of the face. Welcker (1955) considers the condition to be acute seborrhoeic eczema; Malten (personal communication) sees it as a progression from an initial patchy eczema to a later papular and vesicular eczema. Welfling (personal communication) speaks of a " *dermite érythemateuse* " or " *dermite eczématiforme* ". Bory, Lecocq, and Negri (1955) saw 14 cases, some of erythema or urticaria, others of acute vesicular eczema with facial oedema.

Our observations in 11 factories permit the following description:—

The initial stage, lasting from one week to several months, is seen as erythematous itching patches, in the face alone in one in three cases, in the upper extremities in one in five cases, and in the face and upper extremities in one in three cases. Recovery may occur in two to three weeks with scaling. The precise nature of this stage is not understood, but the patches may be a primary irritation due to alkaline hardener or to an early manifestation of sensitization to resin. The effect may never proceed beyond this stage in some operatives, who need not, therefore, cease work, or the process of so-called " hardening " may become established (Schwartz, Tulipan, and Peck, 1947), when the operative becomes sensitized and the second stage appears when contact is resumed. It is characterized by a papulo-vesicular eruption; severe itching leads to scratching, and to the breakdown of vesicles with oozing (wet eczema). The sites of the eruption are as in the initial stage, and the eczematous patches may heal in a few weeks after scaling. Work must be discontinued if this stage is reached. Patch tests show sensitivity to resin in many cases, to hardeners in some and to both in many. Polyvalent sensitization may become established. Table 2 shows the incidence of slight or severe dermatoses

TABLE 2

INVESTIGATIONS OF 11 FACTORIES USING ARALDITE CASTING RESIN D AND HARDENER 951

Factory No.	1	2	3	4	5	6	7	8	9	10	11	Total	Average
Consumption of araldite casting resin D/operative/year (kg.)	381	92	28	62	20	187	100	86	162	31	29	—	—
Total number of operatives handling araldite casting resin D to March, 1955*	84	15	48	15	28	75	4	5	4	32	18	328	—
Total number of dermatoses	56	9	12	7	17	53	3	1	1	2	3	164	—
% of all dermatoses, based on total number of operatives working with araldite casting resin D	67	60	25	47	61	70	75	20	25	6	17	—	43·0
% of slight dermatoses based on total number of operatives working with araldite casting resin D	31	13	8	20	29	29	50	20	25	6	0	—	21·0
% of severe dermatoses based on total number of operatives working with araldite casting resin D (off work for more than three days or permanently)	36	47	17	27	32	41	25	0	0	0	17	—	22·0

*The number of operatives who had worked with "araldite" casting resin D since its introduction could not be established with certainty in all factories; they have therefore in part been estimated by the works management.
Protective measures ranged from poor to very good.

in the 11 factories studied; these correspond closely to the nature of the protective measures used.

The Causes of the Eruptions

From the case histories and our own patch tests it was evident that the skin reactions follow contact with the liquid stage of the resin and hardener or with mixtures of these before setting is complete. This applies both to the stages of apparent direct irritation and of sensitization. A mean time of setting is about 48 hours, before which time contact with the material should be prevented. The set, hardened resin appears to be innocuous as far as skin reactions are concerned. The vapour pressures of the casting resin and of the hardener are so low that atmospheric concentrations are negligible in respect of the early and sensitization effects on the skin of the face, for example. We are of the opinion, therefore, that the problem is one of direct contact of the liquid materials with the skin, but we do not exclude the possibility that even the low concentrations of resin or hardener vapours may be sufficient to induce a recurrence in a sensitized subject.

Preventive Measures

From the nature of the operations in which the liquid materials are used, it is apparent that protection of the worker must be instituted so as to obviate the practices to which the worker himself is prone, *e.g.*, wiping contaminations from the skin with rags impregnated with solvent and resin; wiping perspiration from the face or neck in hot weather with contaminated hands or rags; failure to wash the hands before necessary contacts with the thighs or buttocks.

We consider it necessary to outline in some detail the various aspects of protection, namely, (*a*) instruction to workers and foremen; (*b*) action directed toward the hazard of the contaminated work bench, cleaning rags and solvents, containers; (*c*) removal of vapours by exhaust ventilation; (*d*) protection of the individual skin; (*e*) care of the skin; (*f*) medical measures.

(*a*) The individual worker should be trained by an experienced foreman in how to use these resins and hardeners and taught that traces of casting resin on common articles (tables, chairs, shoes, clothes, machines, buttons, door handles, pencils, etc.) may be the source of skin inflammations. Having regard to the very high incidence of lost time which may result from bad working practice, it is suggested that incentives to care and cleanliness in working should be considered.

(*b*) The work bench and the surrounding area should be covered by a removable layer of strong paper on to which the casting resin may drip to prevent permanent contamination. The soiled, blotched paper could be removed twice daily and destroyed. The degree of soiling of the paper offers a means of assessing the care and efficiency of the workers. Fresh cleaning rags should be supplied daily and used only for occasional cleansing of apparatus, machines, and containers. Contaminated rags should be handled with gloves. The use of solvents for removing spots of resin on the skin or nails should be abandoned. When apparatus is cleaned with solvents, the worker should wear gloves, *e.g.*, of polythene. Ketonic solvents are preferable. Chlorinated and hydrocarbon solvents should be banned. It is desirable that the mixing, distributing and, if possible, casting of the resin should be done with thick, cardboard, spouted containers which can later be discarded, thus avoiding cleansing operations.

(*c*) Although good ventilation and local exhaust at the working point are desirable, especially where sensitized workers are concerned, they are secondary, in general, to the need to prevent direct contact of

the skin with the liquid materials, resin, or hardener.

(*d*) The well-known objection to gloves in work of this kind can to some extent be overcome by the use of soft leather gloves with knitted backs or, alternatively, thin cotton gloves under polythene, since the cured resin mixture does not adhere to the latter. In some diluent operations it may be desirable to use soft leather finger stalls with knitted backs. Whatever method is chosen washing must be thorough and the appliance kept clean and dry ready for use. Barrier creams (" sternocleanse " No. 2, " kerodex " No. 1 or No. 7, " kerodex " BQN 1, " defensyl W ", " fissan " protective ointment) have their place in protection of the skin in this work, especially for the face. Less effective than gloves they nevertheless do offer a certain amount of protection. The use of white overalls and plastic cuffs for the forearms offers additional protection, provided they are washed at least twice weekly, or at once if contamination is visible.

(*e*) For operations of this kind it is desirable to install facilities for washing in the working room, with not too hard brushes and paper towels and barrier creams provided. Malten recommends mild soaps or emulsions or ordinary " sunlight " household soap. In France acid soaps are used. Finger nails should be kept short and clean. After the day's work washing should be followed by inunction of the hands with a mild fatty ointment, *e.g.*, lanoline and vaseline, 1 : 1. The claim of some manufacturers that washing facilities in the workroom have an adverse effect on the processes is not considered justified, since the atmospheric moisture is mainly attributable to the presence of human subjects (perspiration and expiration). The time required for efficient washing and skin protection should be determined and made an official allowance.

(*f*) Pre-employment medical examination should eliminate allergic subjects from work in these processes. If the initial stage of skin irritation develops an attempt can be made to prevent sensitization by reducing the daily period at the bench when the casting resin is used and by intensifying the measures to prevent mechanical irritations and maintained contact with the potential allergens. If such measures fail and sensitization becomes established, further work and contact with the casting resin and hardener must cease. Eruptions should, on the whole, be treated by a dermatologist, only the mildest therapeutic agents being employed in the early stages in the factory.

Summary

Synthetic resins of the ethoxyline group ("araldite" casting resin) have caused many dermatoses. In 11 factories making electrical equipment, the work benches and the workers using " araldite " casting resin D were investigated. One hundred and sixty-four out of 328 workers showed dermatoses which could be classified as skin irritations (erythematous itching patches) and severe allergic eczemas caused by direct contact either with the liquid " araldite resin D " or with hardener 951 (triethylenetetramine), or with a not already polymerized mixture of both.

The preventive measures are discussed in detail. These include (*a*) instruction of workers and foremen; (*b*) action directed towards eliminating the hazard of the contaminated work bench, cleaning rags, solvents, and containers; (*c*) removal of vapours by exhaust ventilation; (*d*) individual protection of the skin; (*e*) care of the skin; and (*f*) medical measures.

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DECOMPRESSION SICKNESS DURING THE SINKING OF A CAISSON

A STUDY OF SOME FACTORS IN THE PATHOGENESIS OF CAISSON DISEASE

BY

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Most of the studies of decompression sickness in industry have been made during the construction of tunnels. The sinking of a caisson, however, is a much briefer undertaking involving certain special features not encountered in tunnel work. Work of this type has received relatively little attention in recent years, although caisson sinking is performed all over the world. This paper describes an investigation into the incidence of decompression sickness during the sinking of reinforced concrete caissons in the Thames between February and May, 1950.

The engineering problem was to build aqueducts to carry river water to a power station for cooling the turbines. The heated water had to be discharged by another outlet aqueduct down stream, well away from the inlet, so that the cooling efficiency would not be reduced by sucking in water already warmed by the turbines. The aqueducts were about 7 ft. in diameter. There were two stages in the construction of each aqueduct: first, caissons were sunk just below low water line in the river; tunnels were then driven out to join them. The aqueduct was completed so that its opening was well above the silt of the river bed. During the sinking the earth was removed by buckets and hoisted up through a " muck lock ". The air pressure in the caisson varied according to its depth below water-level, and was maintained to prevent water or mud rising into the caisson before it was sealed with concrete. The maximum pressure used was about 35 lb./sq.in., though it varied by 10 lb./sq.in. or more with the tide (Fig. 2). The working shift (usually four or five men, sometimes as many as eight) entered and left the caisson by a blister lock in which compression and decompression took place. They reached the caisson bottom by means of a vertical ladder. There were usually three eight-hour shifts. (See Fig. 1 for a diagram of the caisson.)

Methods

Data about cases of decompression sickness were obtained principally from the medical orderlies; they were always on the spot and were responsible for supervising the recompression in the medical lock. Records of all compressions were supplied by the engineer's office.

Measurements were made of the carbon dioxide concentrations in the air in the blister lock of the caisson, during and at the end of a normal decompression. Since this lock is completely filled when it contains five men, there would have been no room for an observer and gas analysis apparatus in it. Accordingly arrangements were made to attach a length of hose to the outlet pipe, from which samples of the air escaping from the chamber during decompression were taken and sealed over mercury. It was thus possible to get estimates of the composition of the air in the decompression chamber while it was being used in the normal way, fully packed with men coming off shift. When a sample was taken another observer recorded the gauge pressure at the time. The oxygen and carbon dioxide were estimated by means of a Haldane gas analysis apparatus.

Results

The general course of the undertaking is shown in Fig. 2, in which are represented the mean weekly pressures under which the work was done, and the weekly incidence of bends. The main part of the figure is concerned with the inlet caisson; the right side of it includes the outlet caisson as well. In the middle of the undertaking work ceased for a period of three weeks. Over the whole period in which the pressure exceeded 18 lb. per sq. in. there were 79 cases of bends affecting 37 men in a total number of 1,800 compressions at the inlet caisson; at the outlet

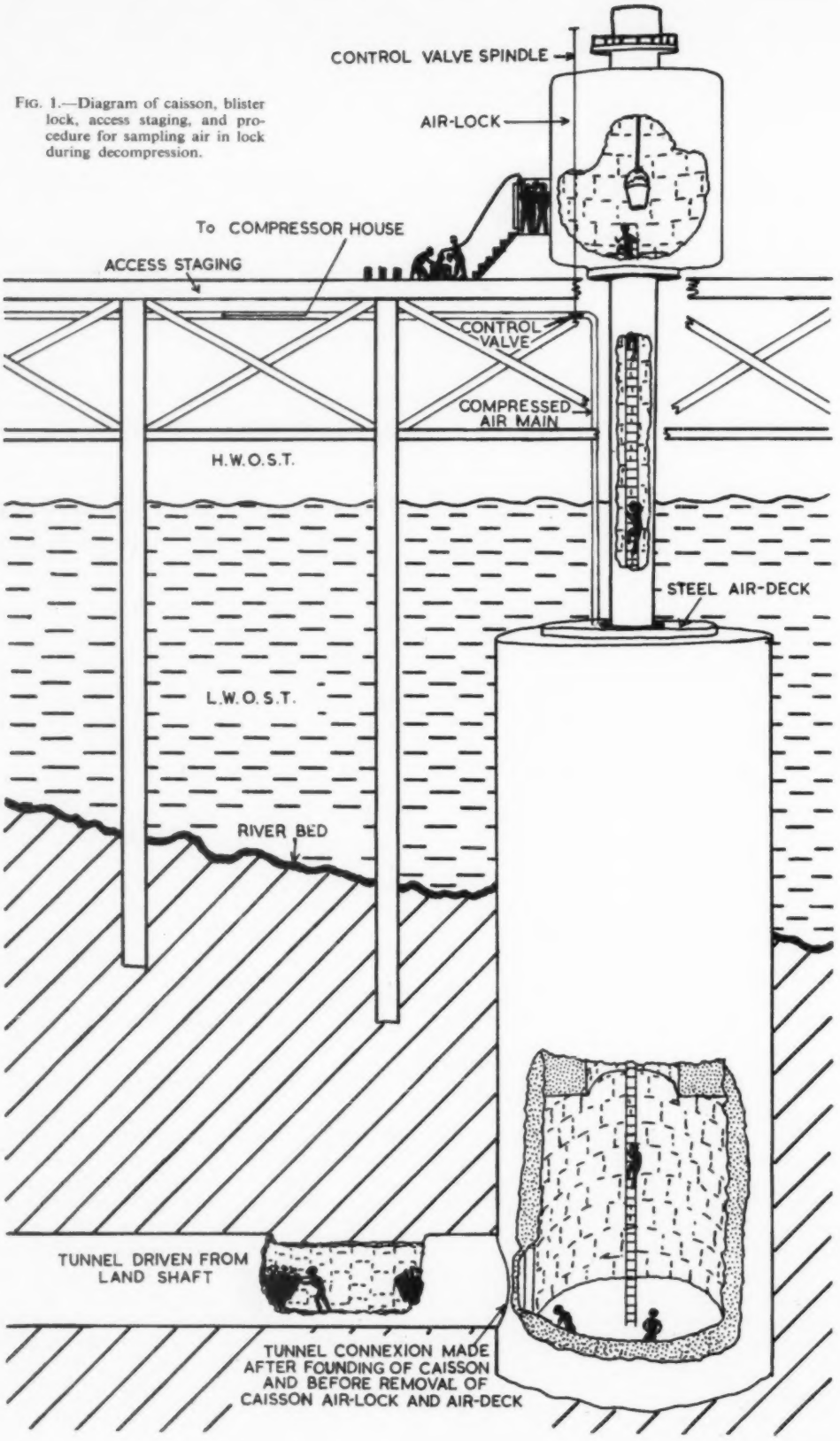

FIG. 1.—Diagram of caisson, blister lock, access staging, and procedure for sampling air in lock during decompression.

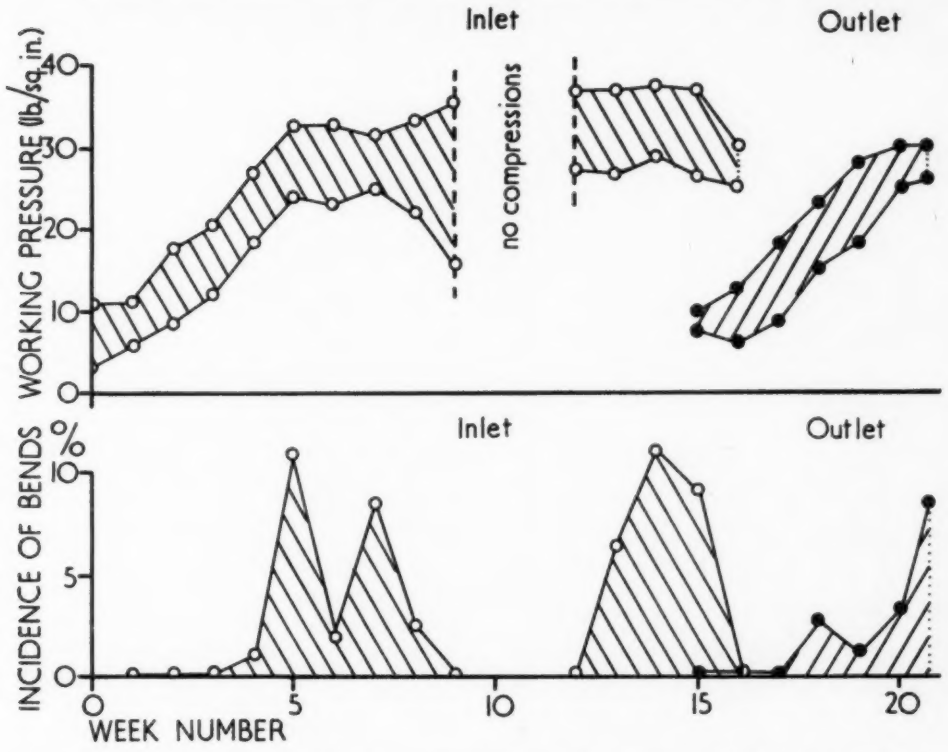

FIG. 2.—Weekly incidence of bends (as % of all compressions) and working pressures (lb./sq.in.). Open circles, inlet caisson, filled circles, outlet caisson.

caisson there were another 10 cases in 310 decompressions. This gives an overall bends rate of about 4%. As is almost always observed in this type of undertaking, the bends rate was far from uniform over the whole course of the work. It only began to rise when the pressure exceeded 20 lb. then rose to a high level for a brief period, and tended to fall away thereafter, even though the pressure was maintained. This almost "epidemic" character of decompression sickness is a very common feature.

The bends, as is usual, consisted of aching pains most commonly affecting the joints. The legs were the most frequently affected, and next the arms; also affected were the knees, wrists, shoulders, head and the body in general. The bends were usually bilateral. The distribution of these symptoms is shown in Fig. 3.

There was the usual tendency for a few individuals to be repeatedly victims of mild bends, and two men had as many as six attacks each. Table I shows how the incidence was distributed.

In view of the rather high incidence of bends, particularly in the shift workers at special times, an attempt was made to define whether there were any special circumstances predisposing to decompression sickness. Four conditions seemed to present themselves:—

TABLE I
FREQUENCY OF ATTACKS OF BENDS IN DIFFERENT INDIVIDUALS

Number of Attacks per Man	Number of Men
1	18
2	10
3	2
4	2
5	3
6	2

(1) **Faulty Decompression.**—When accuracy of decompression was investigated, it was found that the decompression times were not always strictly adhered to. The decompression shown in Fig. 4a, performed during the test for vitiation of the air in the decompression lock, serves as an example. The lock pressure should have been lowered in about two minutes from 27 lb. to 6 lb. per square inch, and thereafter brought to atmospheric pressure during a further 30 minutes. In fact, the pressure fell nearly uniformly throughout, in a time slightly shorter than the total laid down for the decompression.

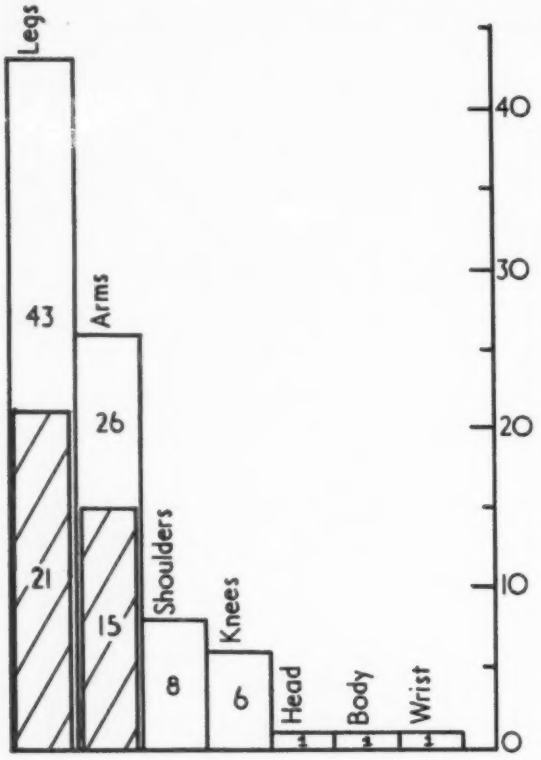

FIG. 3.—Frequency of bends in various parts of the body (hatched columns indicate bilateral involvement).

(2) **Vitiation of Air by Carbon Dioxide during Decompression.**—Also shown in Fig. 4b is the rise of carbon dioxide percentage during the progress of the decompression. It has also been drawn in terms of the absolute tension of carbon dioxide, since it is this that is physiologically important. The absolute tension reached a maximum of 2·63% atmosphere after the decompression had been proceeding for 18 minutes. Thereafter it fell, although the percentage of carbon dioxide in the air in the lock continued to rise. The differing time course of the two measures of carbon dioxide is due, of course, to the changing lock pressure.

It is possible to make an approximate calculation from these figures of the total carbon dioxide production during the period of decompression. In doing this, it is necessary to include in the calculation the carbon dioxide lost in the gas vented from the chamber. When this calculation was made, knowing the capacity of the chamber to be 69 cu.ft., and allowing about 10 cu.ft. for the volume occupied by the five men themselves, we obtained a carbon dioxide production of approximately 0·5 l. per minute, corresponding to an oxygen consumption of about 0·6 l.

(3) **Exercise.**—It was found during the last war that vigorous exercise was a factor precipitating decompression sickness in aviators (Cook, 1951). The evidence for this is much weaker for decompression sickness resulting from exposure to high pressures, but it remains a possibility. It has been noticed in diving experience, for instance, that bends tend to occur in those muscles which have been most heavily worked during exercise. We believe it to be significant, therefore, that at the end of the shift the men had to climb a 70 ft. vertical ladder in order to reach the decompression lock. This ascent was usually made as fast as possible, and there was no waiting period before decompression began. Thus the decompression took place immediately after severe exercise, and this exercise may well be an important contributory factor to the incidence of bends.

(4) **Chilling.**—During the work at the bottom of the caisson the men are usually exposed to considerable heat. This is particularly the case on a warm day, or when concreting is done, since the setting of concrete is an exothermic reaction. On the other hand, when they come into the decompression chamber, the fall in pressure during decompression is associated with a considerable cooling of the atmosphere in which they are placed. This means that not only is the air around them cold and humid, but there is considerable condensation of moisture on their clothes, so that these, already damp, may become thoroughly wet and chilly. Experienced workers sometimes keep a set of dry clothes in the decompression chamber into which they change while waiting. The medical orderlies recorded the impression that men who were not careful about dry clothes or who found the decompression uncomfortably cold were those who seemed to be liable to decompression sickness.

Discussion

In many respects the decompression sickness observed during this undertaking resembled that seen during the construction of a tunnel (Paton and Walder, 1954). Thus it had an " epidemic " character; individuals varied considerably in their susceptibility; and the incidence in different parts of the body was more or less the same as that observed in tunnel work. On the other hand, the incidence was somewhat higher than has been recently recorded in tunnel work. It is probably important to recognize certain differences between the two types of undertaking. In the first place, the construction of a

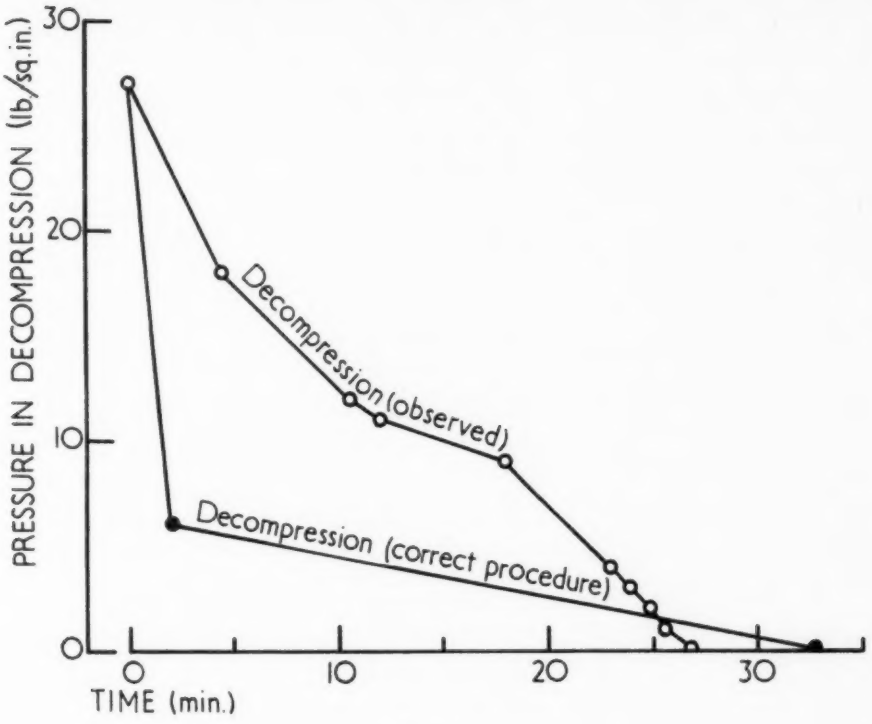

FIG. 4(*a*).—Time course of fall of pressure in blister decompression lock actually observed (open circles) and prescribed by decompression tables (filled circles).

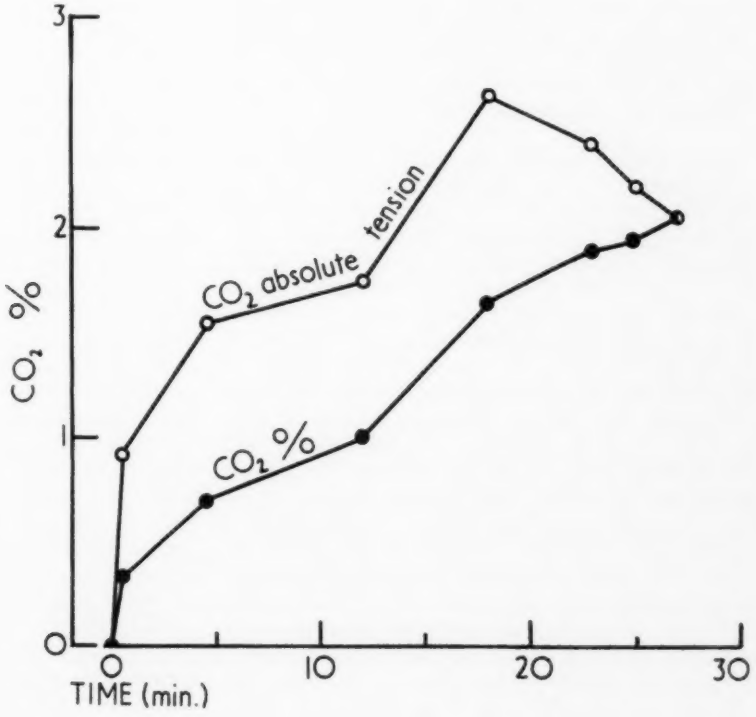

FIG. 4(*b*).—Carbon dioxide percentage (filled circles) and absolute tension in % atmosphere (open circles), during the decompression of five men shown in Fig. 4(*a*).

tunnel is usually a fairly lengthy operation. This allows time for the labour force to acclimatize to the work, for susceptibles to be weeded out, and for all the local details of an efficient decompression procedure to be developed. On the other hand, tunnel work may be more continuous and arduous (at least at the working face) than in a caisson, where there are pauses for the hopper to be hauled to the muck-lock, emptied, and returned. One gains the impression from veteran compressed air workers that bends used to be less of a problem in caisson work than in tunnel work; this might well be due to the existence, before the war, of a useful group of experienced and resistant workers developed by the various tunnel contracts of that time, for whom caisson construction would present no novelty. But with the suspension of tunnel-making during and immediately after the war, this cadre of experienced personnel must have disappeared. One would expect, therefore, that for some years the average bends incidence in short-term compressed air undertakings would be higher than normal, simply because there is no pool of relatively resistant individuals on which to draw.

The absence of a pool of insusceptible individuals does not, however, account satisfactorily for bends rates in excess of 2%. Decompression tables are intended for general use, and should be suitable even with inexperienced workers. For this reason, we have considered whether there are any processes involved in caisson sinking which might make the worker under these conditions relatively vulnerable. Four possible factors emerged.

(1) **Inaccuracy in Decompression.**—The first was simple inaccuracy in decompression. This is a perennial difficulty with all compressed air working, and the chances of securing a sufficiently experienced, competent, and authoritative lock-keeper are usually fairly small, particularly for a relatively brief contract. He has to have a thorough understanding of his responsibility and the rationale of the procedures; but he also needs a personality strong enough not to be influenced by those inside the lock. It is not infrequent, for instance, that eagerness to go to a football match, or to collect and spend the week's pay, leads to a severe outbreak of bends. To prevent over-rapid decompression, the best safeguard is to have a procedure which lays down a definite decompression procedure out of control of the lock-keeper, such as that used in Newcastle, where fixed jets were attached to the outlet pipe. It is comparatively easy to install this system in a tunnel where there is a relatively constant working pressure, but it would be more complicated for caisson work where the pressure is always altering.

A safeguard of a different type is a recording barograph attached to the chamber and secured by lock and key, which provides a permanent record of all the decompressions conducted. This enables examination of decompression procedures at leisure, and permits their correlation with any bends which subsequently occurred.

(2) **Vitiation of Air.**—Vitiation of the air with carbon dioxide during the decompression in the small blister lock appears to be a contributory factor not hitherto recognized. It is known that carbon dioxide can contribute to the genesis of decompression sickness (Cook, 1951). It probably does this by assisting the initiation of the bubble which then grows by the later ingress of nitrogen from the supersaturated tissues. Usually, in tunnel work or in diving or aviation experience, the chambers are of such a size that there is no risk of accumulation of carbon dioxide. The blister lock of a caisson, however, is so small in relation to the number of men crammed into it that significant amounts of carbon dioxide can accumulate. In our observations the highest concentration achieved was about 2·6% of an atmosphere.

But the further question arises as to what this carbon dioxide percentage would have been if the decompression had been properly conducted, instead of having been, as was the case, too slowly falling in the early stages. This is a difficult problem to deal with precisely, since the situation is one whereby the amount of carbon dioxide lost from the blister depends simultaneously on the rate of carbon dioxide production, and on the rate of pressure fall. But an approximation can be obtained quite simply under the present circumstances. In general, if the pressure falls by an escape of the contents of the chamber, instantaneously to (say) half the initial absolute pressure, then the tension of any gas in the mixture of gases comprising the chamber's atmosphere will also fall by half. Similarly if the absolute pressure falls to $x\%$ of its original absolute pressure then the tension of carbon dioxide will fall to $x\%$ of the original value. Now suppose that a major pressure fall takes place right at the beginning of decompression. In this particular case, suppose that the decompression was correctly done and the pressure fell within two minutes from 27 lb. to 6 lb. Now at this period CO_2 can hardly have accumulated; thus virtually no carbon dioxide will be lost from the chamber. The only venting of carbon dioxide will be that achieved later in the further fall of pressure by 6 lb. over 30 minutes. What effect will this gradual loss have on the final carbon dioxide tension? It can be roughly estimated as producing a result halfway between the effects of two extreme

procedures, *viz.*, a fall of 6 lb. rapidly at the beginning of the 30 minutes' period, which could not lead to the loss of any CO_2 and a fall of 6 lb. rapidly at the end of the 30-minute period, which would lead to a maximum loss of CO_2 (from 3·9% to $\frac{15}{15+6} \times 3{\cdot}9$ = 2·8%). This yields a mean figure of $\frac{3{\cdot}9 + 2{\cdot}8}{2}$ = 3·35% CO_2 as the expected final carbon dioxide concentration at the end of a correctly conducted decompression.

It is of interest that a badly performed decompression, such as in this particular case, should actually have lessened the carbon dioxide accumulation in the lock. This happened simply because the major fall of pressure in the lock came later than it should have done after an appreciable concentration of carbon dioxide had accumulated. It is in no way a justification for changing the decompression procedure from that already laid down and well tested.

To overcome this accumulation of carbon dioxide four measures could be taken, two of them easily. (*a*) The men could pause for about 10 minutes after the strenuous climb up the ladder, to allow their metabolism to sink back to normal levels. This should help to reduce their carbon dioxide output while confined in the decompression lock. (*b*) During the decompression the air in the main lock could be flushed out. One way of doing this would be, for instance, to arrange a continuous " bleed " of air into the lock, compensated by having the main outlet slightly more open than usual. It would be technically difficult, however, to achieve a sufficiently large turnover of air to prevent significant carbon dioxide accumulation. Another procedure would be to raise the pressure by a few pounds every quarter of an hour, and then blow it off immediately. The men would probably feel the effects of these changes of pressure, and would have to be warned accordingly. This procedure, too, would be of limited ventilatory efficiency; if the pressure changes were limited to 5 lb./sq.in., it would not change the air more than once during the decompression. (*c*) " Decanting " is a third measure to be considered. Here, the men leave the blister after a very rapid decompression lasting one to two minutes, and immediately go into a more comfortable chamber where they are very rapidly compressed again and then decompressed at the rate laid down. This method would be hazardous if there were any possibility of delay before entering the comfortable chamber. (*d*) If further study showed this to be a serious problem one could easily install a compact blower, operated perhaps by compressed air, circulating the air through a soda lime system. It might, however, be a fairly elaborate undertaking to secure sufficient clearance of carbon dioxide.

(3) **Influence of Physical Work.**—The influence on the incidence of decompression sickness of the physical work done in making the rapid ascent immediately before decompression probably manifests itself by creating nuclei for the formation of bends in the men's muscles. The work of E. N. Harvey and his colleagues (Harvey, 1951) has shown that bubbles do not form in supersaturated solutions unless there is a centre from which the bubble may grow, possibly of the nature of a minute, gas-filled hydrophobic crevice. Damage, or even simple contraction of muscles, is effective in creating nuclei. If practicable, it would be desirable to have a compulsory rest of 10 minutes before entering the lock.

(4) **Chilling.**—Finally, the chilling of the men is probably a factor more prominent in caisson work than in any other type, simply because the temperature at the working face of a caisson reaches such high levels. At the top of the caisson itself room is so cramped that it seems difficult to insist on there being dry clothing available for all workers before they enter the blister. But some form of heating installation is in fact recommended by the Institution of Civil Engineers, and this should be insisted upon.

The enumeration of these factors is not intended as any criticism of those who sink caissons. The difficulties to be surmounted in such undertakings are considerable and much more work on the medical problems involved is needed. But it is hoped that, by indicating what seem to be the likely causes of disability in this type of work, it may help to reduce the incidence of sickness to levels which allow the working force to function at full efficiency without discomfort and without the risk of consequent serious damage to health.

Summary and Conclusions

This paper describes some of the factors in the pathogenesis of decompression sickness during the sinking of a caisson.

Over a period of three months, there were 89 cases of bends, in a total of about 2,100 decompressions.

Among the contributory factors of what was sometimes a rather high rate of decompression sickness were: (*a*) inaccuracy in following the decompression procedure; (*b*) accumulation of carbon dioxide in the blister lock in which decompression took place; (*c*) vigorous muscular exercise immediately before the decompression, due to the ascent of a 70-ft. ladder; (*d*) chilling because of the considerable temperature difference between the hot

working face and the cold decompression chamber.

It is recommended: (1) That lock-keepers should be properly trained and given sufficient authority for their task, that a recording barograph should be attached to any chamber in which decompression of men takes place; (2) that the blister lock should be ventilated or " cleared " during decompression, and that the men should wait for 10 minutes after climbing the caisson ladder before entering it so that their carbon dioxide production should have sunk to resting levels. This waiting period will also tend to reduce the effect of exercise in predisposing to bends; (3) if possible, dry clothes should be provided before entering the decompression lock, and the lock should be kept warm during decompression.

We wish to thank the contractor's engineer for his assistance.

During this work W. D. M. Paton was a member of the Medical Research Council's scientific staff at the National Institute for Medical Research.

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THE OCTOBER (1956) ISSUE

The October (1956) issue contains the following papers:—

The Prevalence of Coalworkers' Pneumoconiosis. Its Measurement and Significance. By A. L. Cochrane, I. Davies, P. J. Chapman, and S. Rae.

Pneumoconiosis of Kaolin Workers. By L. W. Hale, J. Gough, E. J. King, and G. Nagelschmidt.

Carcinoma of the Lung in Workmen in the Chromates-producing Industry in Great Britain. By P. Lesley Bidstrup and R. A. M. Case.

Cancer in Coking Plant Workers. By D. D. Reid and Carol Buck.

The Cytological Diagnosis of Bladder Tumours Amongst Dyestuff Workers. By J. G. S. Crabbe, W. S. Cresdee, T. S. Scott, and M. H. C. Williams.

The White Cell Count and Exposure to Radiation Hazards. By Frances M. Turner.

Dermatitis in Coal-miners. By Arthur Rook and Geoffrey Hodgson.

Miscellanea:

Occupational Mortality: Coronary Heart Disease. By J. N. Morris.

Exposure to Trichloro-ethylene During an Industrial Degreasing Operation. By D. E. Hickish, J. H. Smith, and Joan Bedford.

The British Occupational Hygiene Society: First Provincial Meeting.

The Pneumoconiosis Problem in Coal-mines. By C. G. Warner.

Chlorine: Its Products and Uses. By H. Pirie.

Particle Measurement in Industrial Hygiene. By G. L. Fairs.

Control of the Fluorine Hazard. By J. D. Paterson.

Book Reviews.

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THE EFFECTS OF THE TEMPERATURES OF THE FLOOR SURFACE AND OF THE AIR ON THERMAL SENSATIONS AND THE SKIN TEMPERATURE OF THE FEET

BY

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A century ago the Commissioners who reported to the General Board of Health (1857) on the warming and ventilation of dwellings emphasized that in a comfortable and healthy apartment the floor should be at the highest temperature in the room. This requirement is met in rooms where the floor is warmed. Floor heating is used in Liverpool Cathedral, and Widdows (1921) applied floor heating in schools which he designed for the Derbyshire County Council. More recently the method has been used in a variety of commercial buildings and in houses. This method of heating has the great advantage of giving considerable freedom in planning, and furthermore, no heating apparatus is visible.

Floor heating must be applied with care, however, for if the floor is too hot the feet of the occupants may be overheated so that serious discomfort ensues. This is self-evident, but there seems to be much diversity of opinion concerning the limiting temperature of the surface of a warmed floor beyond which discomfort is likely to arise and limiting values quoted by different authorities vary between 75° and 85°F. It should be mentioned, also, that a risk of discomfort from overheated floors may arise in buildings warmed by means of ceiling panels, for in some such buildings the leakage of heat through the floors is considerable. Thus in a hospital so warmed nurses experienced discomfort from warmed floors. It seems that the risk of discomfort with a given floor temperature will probably vary with the posture and activity of the occupants, but information as to the magnitude of the risk of discomfort under given conditions has not hitherto been available for the guidance of engineers who are designing heating installations.

The present paper describes experiments in which persons were exposed to warmed floors. During the experiments the sensations of warmth of parts of the foot were assessed and the skin temperatures of those parts were measured. The relation between skin temperature and warmth of the foot was then determined. The effects of both the floor surface temperature and the air temperature on (*a*) the skin temperature of the foot and (*b*) sensations of warmth of the foot were then obtained. The percentage incidences of discomfort derived from numerical assessments of the warmth of the feet were transformed to a probit scale for purposes of analysis. The experimental subjects were men and women who in different experiments sat or walked about. From the results of the experiments it was possible to ascertain the risk of discomfort and hence the maximum desirable temperature of the surface of a heated floor for men or women who were walking and sitting.

Methods

The experiments were conducted in a laboratory 17½ ft. × 24 ft. in plan and 14 ft. high. Mechanical ventilation was not available, but with doors and windows closed the ventilation rate varied between one and a half and three air changes per hour, so that with but few occupants there was an ample supply of fresh air. Control of the air temperature and of the atmospheric humidity was not feasible. During experiments draughts were avoided by keeping doors and windows closed, and the effects of solar radiation through the windows were greatly reduced by large screens of aluminium foil. The temperature of the surface of a small wooden platform 9 ft. long and 5 ft. wide, which comprised the floor, was changed by varying the power supplied to electrical resistance mats fixed to the underside of the boarding.

Measurements.—Although a few preliminary measurements of air speed and atmospheric humidity were made, neither of these variables was measured systematically during the experiments. The air was calm, and moderate

variations in humidity at normal room temperatures have but little effect on sensations of general bodily warmth, and probably none on the localized sensations with which this paper is primarily concerned. Measurements made with a radiation thermopile showed that the mean radiant temperature varied only slightly from the air temperature, so that radiation need not be considered separately here.

The air temperature was measured with copper-constantan thermojunctions situated at head level and at 6 in. above the heated portion of the floor, and similar thermojunctions embedded in the surface of the floor were used for measuring the surface temperature of the heated part of the floor. Throughout the experiments the air temperature averaged about 68°F., but the temperatures recorded in individual experiments varied between 58°F. and 76°F. However, the drift in the air temperature in any one experiment was negligible.

The temperatures of the skin of a subject's foot were measured (*a*) on the ankle, at about 1 in. below the medial malleolus; (*b*) on the sole, on the plantar surface of the base of the big toe; and (*c*) on the dorsum of the proximal end of the big toe. These sites will be referred to as the ankle, sole, and toe respectively. The measurements were made with thermojunctions of 36-gauge copper and constantan wires. The test junctions were attached to the skin by small pieces of thin plastic adhesive tape and as a rule were attached to the left foot. So that the subjects could have freedom of movement, the leads from the junctions attached to the skin were connected to the measuring apparatus only when it was desired to measure skin temperatures, and they were then connected to the appropriate terminals of a switch-box similar to that described by Lewis (1930).

Some measurements of the girth of the instep and of the ankle above the malleoli were made with the aid of mercury-in-rubber strain gauges designed by Whitney (1953). Changes in girth of the instep and the ankle were displayed simultaneously on a two-channel photographic recording galvanometer with a period of 0·2 sec.

Subjects and Experiments.—The subjects, three women and five men, sat or walked about for one hour. They wore their normal shoes and clothing. In the sitting experiments they sat in comfortable chairs with their feet resting on the heated floor, while in the other experiments they walked about slowly on the floor.*

For the numerical assessment of thermal sensations the following scale was used:—

Thermal Sensation	Numerical Mark Assigned
Much too warm	+3
Too warm	+2
Comfortably warm	+1
Comfortable	0
Comfortably cool	−1
Too cool	−2
Much too cool	−3

*An experiment, *i.e.*, the exposure of one subject to a given floor temperature for one hour, did not begin until the subject was thermally comfortable, and it was the sensation of warmth and not the skin temperature that decided when the experiment began. There was room for three subjects on the floor, so that, for a given floor temperature, three experiments could be conducted simultaneously. Care was taken to ensure that all subjects were exposed to nearly the same range of floor temperatures.

The subjects reported their sensations of warmth with reference to (*a*) the head, (*b*) the whole body with the exception of the head and feet, (*c*) the ankle, (*d*) the sole of the foot, and (*e*) the toe. These thermal sensations were assessed and the skin temperatures measured at the beginning, the middle, and the end of the period of exposure. The results at the end of the period of exposure were used for analysis.

Analysis of Results.—When the subjects were exposed to the heated floor the feet, and especially the soles, were liable to become uncomfortably warm, so it was appropriate to correlate the numerical marks assigned to the sensations of warmth referred to the sole of the foot with the temperature of the floor surface. In this way interesting and valuable information about trends could be obtained, but in addition it was important also to examine the frequency with which discomfort occurred at various levels of floor temperature, and for this purpose recourse was had to probit analysis (Finney, 1944, 1947; Emmens, 1948). When the recorded sensation was " too warm " or " much too warm ", *i.e.*, + 2 or + 3 on the numerical scale, discomfort, but not necessarily gross discomfort, was experienced. The percentage frequency of occurrence of such discomfort at each level of floor temperature used was ascertained and these percentages were transformed into the appropriate probability units (probits). The probits were then related to floor surface temperatures and regression equations calculated. Four such equations were derived from which may be calculated the maximum desirable surface temperature of a heated floor for men or women who are sitting or walking.

These methods were also employed in studying the relation between skin temperature of the foot and thermal comfort.

Results

Incidence of Discomfort Related to Floor Temperature.—In Table 1 are set out the constants of the regression equations of the form

$$y = a + bt_f$$

where y is the probit of the incidence of discomfort and t_f is the floor surface temperature in °F.

From the equations it can be calculated that the floor surface temperatures at which the incidence of discomfort would be expected to be 50%, *i.e.*, a probit of 5·0, are, for the women, 93°F. when sitting and 86°F. when walking, while the corresponding temperatures for the men are 90°F. and 84°F. respectively. These results suggest that the activity of the subjects is more important than the nature of the footwear so far as tolerance of a warm floor is concerned, for the differences between comparable temperatures for men and women are distinctly smaller than the differences due to activity in persons of the same sex.

The probit regression line for women subjects when walking is shown in Fig. 1.

When the subjects, both men and women, were

TABLE 1
CONSTANTS OF PROBIT REGRESSION EQUATIONS RELATING INCIDENCE OF DISCOMFORT TO FLOOR SURFACE TEMPERATURE

	Women Subjects		Men Subjects	
	Sitting	Walking	Sitting	Walking
a	− 4·00	− 16·24	− 12·86	− 18·08
b	+ 0·097 ± 0·020	+ 0·248 ± 0·038	+ 0·198 ± 0·041	+ 0·275 ± 0·041
χ^2	7·36	7·50	1·46	4·35
No. of degrees of freedom	10	4	9	7
No. of experiments	254	221	125	255

walking the highest floor temperature at which no discomfort was experienced was 77°F. According to Fig. 1 the probit of the expected percentage incidence of discomfort among women at that temperature is 2·9, and that value represents an incidence of 1·8%. According to the probit regression equation for the men when walking the expected incidence of discomfort is 2·8%.

The probit regression equation for women sitting, the constants of which are shown in Table 1, gives for a floor temperature of 77°F. a probit value which indicates that the expected incidence of discomfort would be 6·3%. Similarly, the probits for men sitting show that with that floor temperature discomfort would be expected in less than 1% of the observations. In fact, during the sitting experiments no discomfort was experienced by the women until the floor temperature exceeded 77°F., or by the men until it exceeded 79°F.

The pressure of the foot on the floor is greater when standing than when sitting, and this firmer contact with the floor results in a decreased resistance to the transmission of heat between floor and foot. In walking there is a similar effect during the time the feet are on the floor. Hence, it might be expected that with a given floor temperature discomfort would occur less frequently in sitting than in walking persons. This was so in these observations, for the floor temperatures corresponding to a 50% incidence of discomfort were substantially higher for the sitting subjects (Fig. 2).

Relation between Skin Temperature and Comfort on Different Parts of the Foot.—Correlation of the sensations of warmth referred to the ankle, sole, and toe with the skin temperatures measured on the respective sites showed that the association between sensation and skin temperature was closest in respect of the sole, hence, in further consideration of skin temperature and its relations, only the temperature of the sole will be used.

The zero-order correlations (r) between the numerical marks assigned to the thermal sensations and the temperature of the skin of the sole were:

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Women sitting	+0·68	240
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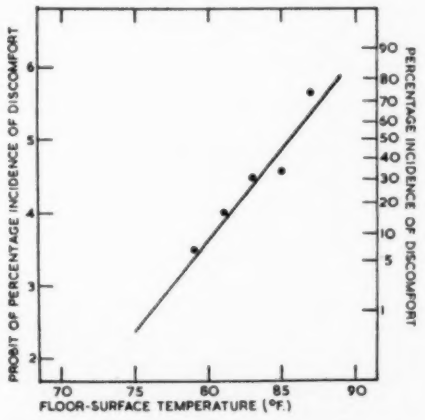

FIG 1.—Probits of the percentage incidence of uncomfortable warming of the soles of the feet in relation to the temperature of the floor surface for women subjects when walking.

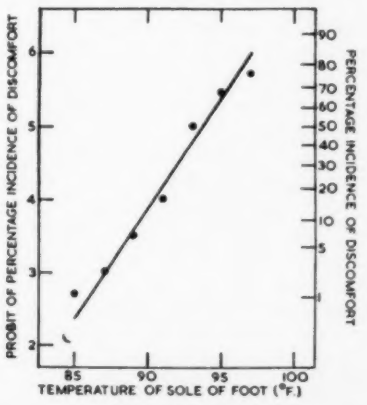

FIG. 2.—The probit of the percentage incidence of discomfort (uncomfortable warming of the sole of the foot after 60 minutes' exposure to a heated floor) plotted against the temperature (°F.) (data from all experiments combined).

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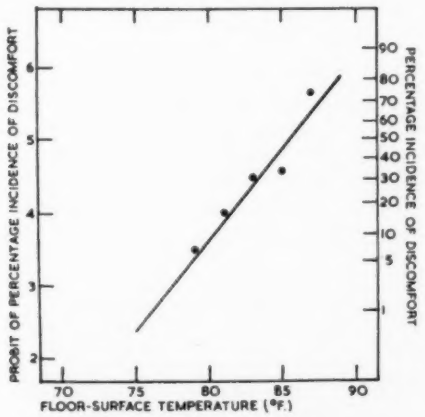

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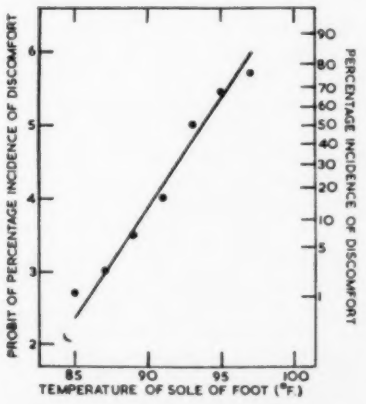

FIG. 2.—The probit of the percentage incidence of discomfort (uncomfortable warming of the sole of the foot after 60 minutes' exposure to a heated floor) plotted against the temperature (°F.) (data from all experiments combined).

The results of the four series of experiments were so similar that it was reasonable to pool the data and to calculate an overall regression for sensation of warmth on skin temperature. The correlation coefficient (r) was $+0{\cdot}72$ ($P < 0{\cdot}001$) and the regression equation

$$S = 0{\cdot}112t_s - 8{\cdot}97 \quad \ldots\ldots\ldots \text{(i)}$$

where S is the numerical value of the sensation, and t_s is the skin temperature on the sole in °F.

Although the value of the correlation coefficient is high, it is not high enough to enable one to predict individual sensations of warmth with useful accuracy.

The relation between the incidence of discomfort through excessive warming of the feet and the skin temperature of the sole was also examined by the probit method. The probit regression equation was

$$P = 0{\cdot}304t_s - 23{\cdot}50 \quad \ldots\ldots \text{(ii)}$$

where P is the probit of the percentage incidence of discomfort and t_s is the skin temperature of the sole in °F. The standard error of the regression constant is $\pm 0{\cdot}023$, and the skin temperature associated with a 50% incidence of discomfort is $93{\cdot}6° \pm 0{\cdot}4$°F.

Relation between Skin Temperature of Sole and Environmental Temperatures.—It seemed reasonable to expect that the temperature of the skin on the sole, which is so closely related to the thermal comfort of the foot, would be affected, not only by the temperature of the floor, but also by the air temperature, and in order to examine this point the skin temperatures measured at the end of an hour's exposure were correlated with the temperatures of the floor and of the air. Table 2 gives the partial coefficients thus obtained together with the total correlation (R) of skin temperature with the two environmental temperatures.

Table 2 shows that on the whole the skin temperature of the sole tended to be more closely associated with the temperature of the floor surface than with the air temperature. The influence of the latter was not negligible, however, as the constants in the partial regression equations make clear. These constants for equations of the form

$$t_s = at_a + bt_f + c$$

where t_s, t_a, and t_f are the temperatures of the skin, the air, and the surface of the floor respectively, are set out in Table 3.

TABLE 3

PARTIAL REGRESSION COEFFICIENTS OF SKIN TEMPERATURE OF THE SOLE ON AIR AND FLOOR-SURFACE TEMPERATURES

Equation for	Partial Regression Coefficient of Skin Temperature on		Added Constant
	Air Temperature	Floor Temperature	
Women sitting	0·494 ± 0·113	0·351 ± 0·039	25·70
,, walking	0·451 ± 0·104	0·488 ± 0·109	18·07
Men sitting	0·603 ± 0·202	0·379 ± 0·121	14·81
,, walking	0·280 ± 0·128	0·813 ± 0·145	0·52

The regression constants show that the air temperature had a greater effect when subjects sat than when they walked, that the temperature of the floor surface exerted a greater effect when subjects walked, and that the effect of a given change in air temperature was greater than that of the same change in floor temperature when the subjects sat and less when they walked. The standard errors of the coefficients show, however, that too much stress should not be laid on these indications, for the differences between the coefficients for air temperature and floor temperature in the four equations could be due to chance, although in the equation for men walking that is not very likely. In each of the first three equations the sum of the two regression coefficients is less than unity, indicating that increments of 1°F. in the temperatures of both the air and the floor surface are accompanied by a rise of rather less than 1°F. in the temperature of the skin of the sole. In the equation for men walking the sum of the coefficients is greater than unity and it is doubtful whether the equation would fit observations made over a range wider than that covered by the present observations.

Relation between Sensations of Warmth on the Sole and Air and Floor Surface Temperatures.—Correlations similar to those just described for the temperature of the skin were also calculated for the sensations of warmth of the sole of the foot after one hour's exposure. The total correlations

TABLE 2

PARTIAL CORRELATION OF SKIN TEMPERATURE OF SOLE OF FOOT WITH AIR AND FLOOR SURFACE TEMPERATURES

Skin Temperature Correlated with	Held Constant	Correlation for Women		Correlation for Men	
		Sitting	Walking	Sitting	Walking
Air temperature	Floor temperature	0·288 ($P < 0{\cdot}001$)	0·292 ($P < 0{\cdot}001$)	0·262 ($P < 0{\cdot}01$)	0·141 ($P < 0{\cdot}05$)
Floor temperature	Air temperature	0·504 ($P < 0{\cdot}001$)	0·299 ($P < 0{\cdot}001$)	0·274 ($P < 0{\cdot}01$)	0·342 ($P < 0{\cdot}001$)
Total correlation (R) of skin temperature with air and floor surface temperatures		0·546	0·674	0·469	0·594
No. of experiments		240	202	121	238

(R) of the numerical marks assigned to the sensations of warmth with the temperatures of the air and of the surface of the floor are given below.

Subjects and Activity	Total Correlation (R) of Sensations of Warmth with Air and Floor Surface Temperatures
Women sitting	0·506
,, walking	0·624
Men sitting	0·431
,, walking	0·494

The total correlations of sensations with the temperatures of the air and of the floor are all lower than the comparable zero-order correlations of sensations with skin temperature reported earlier in this paper. Each of these correlations is rather smaller than the comparable correlation of skin temperature with the environmental measurements shown in Table 2.

The partial regression coefficients for sensations on air temperature and floor surface temperature, which are set out in Table 4, vary in much the same way as the coefficients for skin temperature shown in Table 3.

TABLE 4

PARTIAL REGRESSION COEFFICIENTS OF SENSATIONS OF WARMTH OF SOLE OF FOOT ON AIR AND FLOOR SURFACE TEMPERATURES

Equation for	Partial Regression Coefficient of Skin Temperature on		Added Constant
	Air Temperature	Floor Temperature	
Women sitting	0·071 ± 0·014	0·037 ± 0·005	− 7·19
,, walking	0·055 ± 0·016	0·069 ± 0·020	− 8·48
Men sitting	0·075 ± 0·032	0·060 ± 0·019	− 9·48
,, walking	0·033 ± 0·023	0·124 ± 0·028	−11·39

The earlier remarks concerning the regression of skin temperature on the environmental measurements apply also to the figures in Table 4. Although here also air temperature exerts the greater effect on sitting subjects and the floor temperature on walking subjects, the differences between the regression coefficients are not statistically significant and the regression equations should therefore be used with caution.

Discussion

Maximum Desirable Temperature of a Warmed Floor.—In considering the range of environmental warmth which can be regarded as generally acceptable from the standpoint of general bodily warmth as distinct from the local sensations with which the present paper is concerned, various standards have been adopted by different workers. When comfort investigations are carried out on any considerable number of persons it is commonly found that under no conditions are all the people concerned completely satisfied, and that at the temperature which best suits the greatest number of people some will feel too warm while others will feel too cool. Some workers have taken as acceptable the range of conditions within which sensations of " slightly too warm " to " slightly too cool " were experienced. Others have taken the range within which on a numerical scale of sensations the average value would be " comfortably warm " and " comfortably cool ", while yet others have used the range within which not less than 50% of the votes ranged from " comfortably warm " to " comfortably cool ". It is clear that with the last-mentioned criterion many people will be uncomfortable as the limits of the so-called " comfort zone " are approached and some of the other methods also lead to undesirably wide zones. With these considerations in mind, Bedford (1936) recommended as a practicable comfort zone that range within which, at any temperature, not less than 70% of the persons were quite comfortable. Within that zone at least 86% of the comfort votes recorded ranged from " comfortably cool " to " comfortably warm ", so that never more than 14% of the persons were actually uncomfortable.

While from the standpoint of the general level of warmth it must be accepted that a small proportion of persons may be rather too warm or rather too cool, it is important that the method adopted for warming a room should not be one that is liable needlessly to increase the number of dissatisfied persons by causing local discomfort. In an earlier study of the effects of heated ceilings on comfort (Chrenko, 1953), the experimental subjects reported any feelings of unpleasantness that they experienced while exposed to a heated ceiling. The symptoms of discomfort, usually referred to the head, were variable and very ill-defined. The subjects were asked to report any feelings of unpleasantness, and it may be that in comparison with the usual occupants of an office or other place of work provided with ceiling heating they were hypercritical. However that may be, mildly unpleasant sensations were reported when the intensity of the radiation was very small, and in consequence the recommendations as to the desirable temperatures of heated ceilings of various dimensions were framed on the assumption that, in practice, conditions which caused slight over-warming of the heads of the experimental subjects on 10% of occasions would be acceptable. In connexion with the experiments on heated floors, however, no such compromise appears to be necessary, for a considerable amount of warming can be achieved by heating the floor to a temperature which, according to the results here reported, is unlikely to occasion even mild discomfort.

The maximum floor temperature at which none

of the subjects experienced discomfort in these experiments was 77°F. It should be noted, however, that the probit regression equations (Table 1) suggest that with that floor temperature discomfort might be expected to arise in some 2 or 3% of observations. Furthermore, the experiments were continued for only one hour and it is possible that while the subjects experienced no discomfort with a floor temperature of 77°F. in that time, some of them would have been slightly uncomfortable if they had remained on the heated floor for three or four hours. Hence, while the floor temperature should not exceed 77°F., it would be still better if, except in severe weather, it did not exceed 75°F.

This finding is in close accord with the view expressed earlier by Bedford (1951) in the light of experience in schools with heated floors. It had been noted that when the surface temperature of the floor did not much exceed 75°F. the heating seemed satisfactory, but that if the temperature reached 80°F. the hot floor was tiring to the feet. Hence Bedford concluded that the temperature of a heated floor should not exceed 75° or perhaps 77°F. Liese (1951) expressed the view that 77°F. should be the maximum temperature for a heated floor, and Missenard (1951) and Squassi (1951) allowed a maximum of 82·5°F. These views are based on general experience of floor heating rather than on experiment. On the other hand, Herrington and Lorenzi (1949) conducted a series of experiments in which the thermal sensations of the subjects were noted and regional skin temperatures measured. They concluded that floor temperatures above 75°F. were physiologically undesirable and that the local effect of a hot floor was clearly demonstrable when the floor temperature reached 80°F.

The results for the sitting subjects in this investigation can be compared directly with those obtained by Maréchal (1955), who tested the thermal reactions of 14 women and 17 men when they were sitting. The average air temperature in Maréchal's experiments was 71°F., or three degrees higher than in the experiments here described. Maréchal pooled the results of his experiments on men and women, presumably because of the variety of the shoes worn, especially that of the women's shoes which, he says, defied classification. Fig. 3 shows the percentage incidence of discomfort among the sitting subjects (men and women) in relation to the temperature of the floor surface (*a*) for these experiments, and (*b*) for Maréchal's experiments.

The curves shown in Fig. 3 are derived from the probit regression equations of the incidence of discomfort on floor surface temperature. The equation for Maréchal's results is found to be

$$P = 0{\cdot}148t_f - 8{\cdot}36 \quad \ldots\ldots \quad \text{(iii)}$$

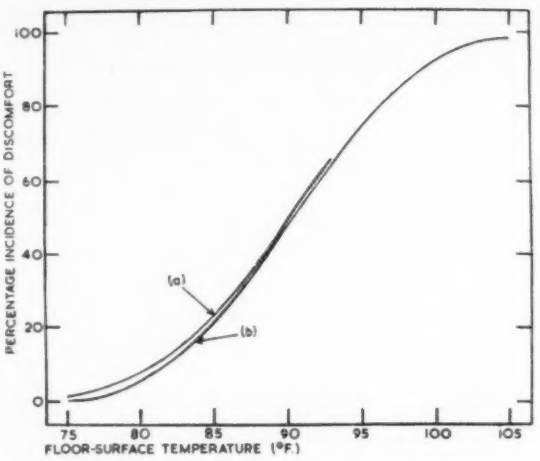

FIG. 3.—The percentage incidence of discomfort in men and women subjects when sitting plotted against the surface temperature of the floor (°F.), curve (*a*) representing the present experiments and curve (*b*) the results of Maréchal (1955).

where P is the probit of the percentage incidence of discomfort and t_f is the temperature of the floor surface in °F. The corresponding equation for the combined results for men and women in the present experiments is

$$P = 0{\cdot}142t_f - 7{\cdot}79 \quad \ldots\ldots \quad \text{(iv)}$$

The two equations are practically identical and the curves shown in the figure are almost congruent.

It has been remarked earlier that the activity of the subjects seems to be more important than the nature of the footwear, so far as tolerance of a warmed floor is concerned. No attempt was made to classify the shoes worn by our subjects. The soles of the men's shoes were much thicker than those of the women's, yet, after examination of a cut section of a man's worn shoe, it was apparent that estimates of the thickness of the sole based on inspection could be very misleading. Accordingly a special calliper was constructed so that the thicknesses of the soles of intact shoes could be measured. Such measurements showed that the difference between the thicknesses of the soles of men's and women's shoes was much less than had been expected, the average thickness of a man's shoe sole being 0·52 in. and that of a woman's 0·38 in. It seems likely that the somewhat greater tolerance of floor heating shown by the men when they were sitting was mainly due to the effect of the medium-weight woollen socks worn by them, whereas the women wore nylon stockings. The decreased thermal resistance of the men's socks under pressure may account for the similarity of the tolerance of floor heating shown by men and women when walking.

Attempts have been made to compute the maximum desirable floor temperature from physical parameters. Fischer (1954) made such calculations, assuming that for comfort the skin temperature on the sole of the foot should not exceed 85°F. with which temperature, according to the present results, the incidence of discomfort would be less than 1%. Fischer concluded that the maximum desirable temperature of the surface of a floor was 77°F. which agrees with our finding.

TABLE 5

ESTIMATED FLOOR-SURFACE TEMPERATURES GIVING SENSATIONS OF WARMTH ON SOLE SIMILAR TO AN AIR TEMPERATURE 68°F. AND FLOOR TEMPERATURE 77°F.

Experimental Group	Estimated Floor Temperature with Air Temperature (°F.) of		
	60	65	70
Women sitting	92	83	75
,, walking	84	79	75
Men sitting	87	81	75
,, walking	79	78	77

Air Temperature and Tolerance of a Warmed Floor.—The average air temperature during the experiments was 68°F. so that the conclusion that the floor temperature should not exceed 77°F., or preferably 75°F., applies to rooms which are maintained at what is commonly regarded in Great Britain as a comfortable indoor temperature. The partial regression coefficients recorded in Tables 3 and 4, however, indicate that the skin temperature and a feeling of warmth on the foot are affected by the temperature of the air as well as by that of the floor. As the air temperature is lowered a higher floor temperature can be tolerated. Although with one exception the partial regression coefficients are statistically significant, the standard errors are rather large, and too much emphasis should not therefore be laid on the values of skin temperature or of warmth sensations calculated from the equations. But it is, nevertheless, of interest to estimate what would be expected to be permissible floor temperatures with air temperatures differing from 68°F.

For the four groups of observations the average sensations of warmth on the sole of the foot are calculated for an air temperature of 68°F. and a floor temperature of 77°F., using the regression constants of Table 4. They do not differ greatly from + 0·5, or midway between exactly "comfortable" and "comfortably warm". Using the sensation values thus ascertained, the floor temperatures which with various air temperatures would be expected to give rise to similar feelings of warmth of the foot have been calculated (Table 5).

Table 5 suggests that in the first three groups a reduction of 10°F. in the air temperature raises the tolerance of a heated floor considerably. Some doubt has already been expressed about the regression constants for men walking.

Missenard (1955a) obtained results which appeared to show increased tolerance of a warmed floor when the air temperature was lowered, but it is difficult to make a detailed comparison of his results with those of the present experiments owing to differences in the methods of experimentation. In a further paper, however, Missenard (1955b) describes an experiment in the basilica of St. Quentin. The heating of that building was inadequate and it was decided to heat an area of 20 sq. metres, or about 215 sq. ft., of the floor. When the air temperature was as low as 41°F. and the heated portion of the floor was at a temperature of 102°F., only 1% of that part of the congregation exposed to the heated floor complained of over-warm feet. From the regression constants in Table 4 one can calculate that for sitting persons wearing ordinary indoor clothing the average sensation of warmth on the soles of the feet under such conditions would be − 0·2 or − 0·5 on our arbitrary scale. This is a gross extrapolation below the range of conditions included in our experiments, but it does appear that when it is not practicable to warm a building to normal standards of comfort severe discomfort from cold feet can be avoided by raising the surface temperature of the floor to a level that would be most undesirable with a higher air temperature.

Changes in Girth of Foot and in Blood Flow.—One criterion for deciding upon the maximum desirable temperature of a floor must be human sensations, and our results show clearly that the sensation of warmth on the sole of the foot is much affected by the temperature of the floor. Yet it is conceivable that a heated floor might exert some other influence of which one was not aware. For instance, it seems possible that when the feet are on a heated floor there may be venous stasis and some swelling of the foot, even though there is no thermal discomfort. An attempt was made to test this. Volume plethysmography was not practicable because it was necessary to carry out the tests under conditions similar to those that prevailed during the main series of experiments. Hence, mercury-in-rubber strain gauges were used to detect any changes in the girths of the instep and ankle.

The results of numerous experiments were indeterminate. In one woman swelling of the feet was detected in a control experiment when the floor was not heated, while no swelling was detected in other subjects when their feet were on a floor heated to an uncomfortably high temperature.

Allwood and Burry (1954) studied the flow of blood through the feet in relation to the temperature of the water in their plethysmograph. The blood flow was measured by the method of venous occlusion. It was found that the flow increased only slightly with temperature up to about 86°F., but at higher temperatures it increased markedly. From the probit regression equation for the incidence of discomfort against skin temperature (equation (ii)) it can be seen that at a skin temperature of about 85°F., which corresponds closely with the 86°F. at which the blood flow rose sharply, there was in these experiments a sharp rise in the incidence of discomfort. It seems likely, therefore, that in our subjects discomfort through over-warming of the feet was associated with a notable increase in the flow of blood through the feet, even though no consistent evidence of swelling was found. It also seems likely that there was no notable physiological change until discomfort began to appear, so that the thermal sensations may be regarded as a reasonable index of physiological effect.

Skin Temperature and Comfort.—It has been noted earlier that when the results of all the experiments were grouped together there was a highly significant correlation between the skin temperature on the sole of the foot and the local sensation of warmth. The average sensations of warmth reported at various skin temperatures fitted closely about the regression line (equation (i)), but, as one would expect, the individual sensations of the members of a group of people cannot be predicted with any useful degree of accuracy. The regression equation of the probits of the percentage incidences of discomfort on various skin temperatures (equation (ii)) fits the results very well, and gives a reliable indication of the proportion of people who will be uncomfortable. Thus, with a skin temperature of 90°F. the expected incidence of discomfort is $14 \pm 5\%$.

A regression equation such as equation (i), which is derived by the method of correlation and in which the dependent variable is the thermal sensation expressed in arbitrary units, enables one to estimate how warm the average person will feel, but it gives no indication of the proportion of people who will be quite comfortable or uncomfortable and this is of importance in considering what should be regarded as limiting conditions. One can, however, arrive at such an indication indirectly by combining equations (i) and (ii) and eliminating t_s. Thus, from the present observations one obtains the equation

$$P = 2{\cdot}71S + 0{\cdot}85 \quad \ldots\ldots\ldots \text{(v)}$$

where S is the mean thermal sensation on the arbitrary scale and P is the probit of the percentage incidence of discomfort.

Probits estimated from this equation have been converted into percentages and these are plotted against sensations in Fig. 4.

With the sensations of local warmth with which this paper is concerned, with an average sensation of + 1·5 or midway between "comfortably warm" and "too warm", the expected incidence of discomfort is about 50%, which agrees with an equation reached by Chrenko (1955) from a consideration of the results obtained by Ellis (1952) in a study of general bodily warmth.

Equation (iii) enables one to estimate the probable incidence of discomfort from estimates of the mean thermal sensations that would be associated with various combinations of air temperature and floor temperature. For the various groups of subjects the constants for the partial regression equations of sensation on air temperature and floor temperature are given in Table 4. If one considers, for example, the women subjects when walking, the calculated mean thermal sensation for an air temperature of 65°F. and a floor temperature of 75°F. is + 0·3 on the arbitrary scale, while with the same air temperature and a floor temperature of 85°F. the sensation is + 1·0. According to equation (v), with these mean sensations the probits of the incidence of discomfort are 1·66 and 3·56 respectively, and according to Fig. 4 the percentage incidence of discomfort with the lower floor temperature would be less than 1%, whereas, with the higher one it would be about 7%.

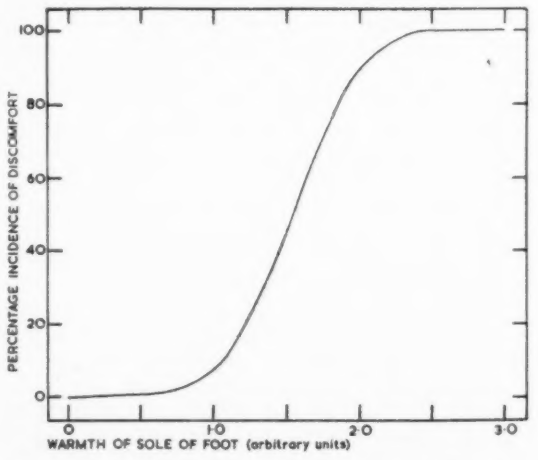

FIG. 4.—The percentage frequency of uncomfortable warming of the sole of the foot plotted against the sensation of the warmth of the sole expressed in arbitrary units. (Data from all experiments combined and warmth of the sole assessed after 60 minutes' exposure to a heated floor.)

Summary

Experiments were made on three women and five men who wore their normal shoes and clothing. For one hour they sat with their feet on a heated floor or walked slowly to and fro. Altogether, 379 experiments were made on sitting subjects and 476 on walking subjects.

The average air temperature was 68°F., but the temperature of different experiments ranged from 58°F. to 76°F.

Discomfort arising from over-heating of the floor was due to excessive warming of the feet, as shown by high skin temperatures, especially on the soles. A skin temperature of 93·6°F. on the sole of the foot was associated with a 50% incidence of discomfort. A regression equation relating the probable incidence of discomfort to skin temperature is given.

No uncomfortable warming of the feet occurred until the floor temperature exceeded 77°F., and it is concluded that in a room maintained at a normal temperature the floor temperature should not exceed 77°F., or, better, 75°F.

The temperature of the air affects the tolerance of a warm floor, so that a higher floor temperature can be tolerated as the air temperature is lowered. Regression equations are given which relate the average sensation of warmth (expressed on an arbitrary numerical scale) to the temperature of the air and floor.

Mercury-in-rubber strain gauges were used to detect any changes in the girths of the instep and ankle when subjects were exposed to a heated floor. The results were indeterminate. Swelling occurred in one subject when the floor was not heated, while with other subjects there was no swelling when the floor was uncomfortably hot.

The temperature of the skin of the sole at which the incidence of discomfort began to rise rapidly was about the same as that at which a rapid increase in blood flow might be expected.

The results of the present experiments are compared with the conclusions reached in some earlier investigations.

The author's thanks are due to Dr. T. Bedford, Director of the Environmental Hygiene Research Unit, for his advice and helpful criticism during the course of the work, and to all those members of the Unit who kindly volunteered to act as subjects. Especially thanks are due to the Misses R. J. Pearse and B. E. Tredre, who not only acted as subjects, but also gave valuable assistance during the analysis of the results, to Mr. F. E. Smith, M.B.E., for designing and constructing the special calliper used in the experiments, to Dr. R. J. Whitney, who gave advice on the construction and use of his strain gauges, and to Mr. C. R. Underwood, who constructed the bridge used in the experiments with the strain gauges.

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FOOT RINGWORM IN COAL-MINERS*

BY

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Ringworm of the feet was first reported in Britain by Whitfield (1908). It leads to irritation and to various eczematous manifestations of and about the toes and sometimes elsewhere on the skin of the feet: the nails may also be affected. The common causative fungi are *Trichophyton mentagrophytes*, *T. rubrum*, and *Epidermophyton floccosum*. This paper describes an epidemiological study of the disease as it affects industrial workers, especially coal-miners.

By 1951 it was clear that ringworm was a troublesome complaint in coal-miners. A statistical study of compensation data carried out at the Headquarters of the National Coal Board had suggested that pithead baths were important factors. This fact was capable of several interpretations:—(1) That baths favoured the spread of ringworm (Memmesheimer, 1936; Knowles, 1943; Adamson and Annan, 1949); (2) that bathing so altered the immunity of the host as to make infection with the parasite possible; (3) that bathing aggravated pre-existing infection (Riddell, 1951); (4) that factors associated with bathing—hot working conditions in deep hot pits with baths—were the cause of the aggravation or change in the host-parasite relationship.

Apart from the site, nature, and condition of a man's employment, various factors common to mineworkers and non-mineworkers had also to be considered, namely, age and seasonal variation and geographical variation throughout the different parts of Great Britain. It was decided that a field survey was the only way to obtain the information required.

The Pilot Survey

A pilot survey was carried out at Pit A, a colliery with baths. With the aid of tables (Kendall and Babington Smith, 1939) a random sample of 200 men was selected from the nominal rolls; 195 were examined.

As a result of this pilot study, we concluded: (1) That the men would respond readily, provided there was a system of appointments and the examination took no longer than five minutes; (2) that the methods used for clinical examination and laboratory diagnosis were satisfactory; (3) that body ringworm was rare and unimportant; (4) that foot ringworm was common. The clinical diagnosis was difficult, and laboratory confirmation was essential.

It was decided to limit further work to a study of foot ringworm only.

The Survey Proper

Method.—It was not possible to select the places for survey strictly at random, but it was arranged that a wide geographical area was covered. Visits were made to 11 pits, a training centre, and two power stations; the location of each is shown in Fig. 1.

In contrast to the selection of pits and power stations, the men were selected strictly at random as in the pilot study. Since separate nominal rolls were kept for men doing different work, it was possible to allot quotas to the various occupational groups and to increase the quotas of the groups which interested us most.

As far as possible the procedure at the interview was the same for each patient. After a standard history and clinical examination, at least one skin sample was taken from each foot. The only variations were (1) that help was sometimes available for

*This work was carried out under the direction of the Committee for Industrial Epidermophytosis of the Medical Research Council, Dr. J. T. Ingram (Chairman), Dr. J. B. Adamson (died June, 1955), Dr. G. C. Ainsworth, Dr. A. L. Cochrane, Dr. T. E. Howell (resigned owing to ill-health, 1953; died June, 1954), Professor G. H. Percival, Dr. D. D. Reid, Dr. J. M. Rogan, Dr. H. R. Vickers, Dr. B. S. Lush (Secretary). Reports to the Committee containing full details of the work have been deposited in the library of the National Institute for Medical Research, Mill Hill, N.W.7, and may be consulted on application. The clinical data formed part of a thesis by one of us (J. G. H.) and a précis of this paper was read by him at the Annual Meeting of the British Association of Dermatology in July, 1955.

FIG. 1.—Location of places surveyed.

the taking of the history and (2) that additional skin and/or nail specimens were sometimes taken from men with suspicious lesions.

The skin and nail samples were posted to the mycologist in numbered slips of paper. He processed the day's specimens in any order and gave the same treatment to each. Twelve fragments each of about 1 mm. in diameter were cultured on 4% malt extract agar at 28° C for three weeks; microscopic examination was made after clearing in 20% KOH. The origin of the material was not known in the laboratory until both microscopical and cultural results had been reported. Mosaic (Weidman, 1927) was not considered as evidence of infection.

Results

Response Rate.—Of the 2,160 men selected for examination, 2,101 (97%) were seen—a very satisfactory response. Of the remaining 3%, one-third refused and two-thirds were not available for unavoidable reasons, such as being in hospital. As far as could be ascertained, none of those off sick were suffering from a skin complaint.

Main Clinical and Laboratory Findings.—Ninety per cent (1,900 out of 2,101) of the men examined had some clinical abnormality of the skin of the feet, which would have called for laboratory examination in routine clinical practice. By contrast the mycologist found only 21% (438) to be infected. As five of the infected men had feet which were clinically normal, the proportion of men with lesions who were proved to be infected was 23%. (Details are given in a separate publication (Holmes and Gentles, 1956).)

Detailed Laboratory Findings.*—The number of men infected was 438 (21%). The grounds for diagnosis of the 438 infected men were: culture and microscopy, 346, culture only, 57, microscopy only, 32, others, three (two *Candida albicans* and one doubtful). The cultural results were: *T. mentagrophytes* 224 (all but two of the interdigitale strain), *T. rubrum* 148, *E. floccosum* 11, *T. mentagrophytes* and *T. rubrum* 13, *E. floccosum* and *T. rubrum* 4, *E. floccosum* and *T. mentagrophytes* 3.

On the occasions when two dermatophytes were obtained from a patient they were isolated from the same lesion (combined infection) 11 times and nine times from different sites (concurrent infection). Whilst the overall species distribution was *T. mentagrophytes* 61%, *T. rubrum* 35%, and *E. floccosum* 4%, in the multiple infections the proportions were 80%, 85%, and 35% respectively. Hopkins, Hillegas, Ledin, Rebell, and Camp (1947) have noted this relatively high frequency of *E. floccosum* and Partridge (1955) discusses in detail the presence of *T. rubrum* in multiple infections.

The Effect of Environment

Geographical Variation.—The prevalence rates as proved by the laboratory findings varied from 3·5% to 50%; at the same time there was some grouping (Table 1). The two pits in Scotland (F and G) had similar rates as had the two power stations in London (N and O), but other results could not be

TABLE 1
PREVALENCE RATES FOR INDIVIDUAL PLACES VISITED

Place	No. of Observations	% with Positive Laboratory Report
B	200	3·5
C	217	50
D	149	32
E	153	31
F	72	16
G	74	17
H	150	21
I	146	6
J	221	28
K	169	8
L	148	15
M	124	12
N	68	25
O	62	26
B¹	148	10
Total	2,101	21

*Cultures and herbarium specimens of the three species of dermatophytes have been deposited at the Centraal bureau voor Schimmelcultures, Baarn, and at the Mycological Reference Laboratory The London School of Hygiene and Tropical Medicine, W.C.1.

explained on geographical grounds. Neighbouring places had widely different rates, *e.g.*, C 50%, M 12%, and similar rates were obtained from different parts of the country, *e.g.*, D 32%, J 28%. It was therefore necessary to look for other factors which might have been influencing the rates, *viz.*, the presence of pit-head baths, conditions underground.

The General Effect of Communal Baths.—Four pits (I, K, L, M) had no pit-head baths; the prevalence rate ranged from 6% at I to 15% at L. At the pits and power stations with baths (C, D, E, F, G, H, J, N, O) the rates ranged from 16% at F to 50% at C. Pit B was visited twice—nine months before and six months after the completion of pit-head baths; the rates were 3·5% and 10% respectively.

Not all the men at the pits with baths used them and at two pits without baths, L and M, there were showers for officials.

In the tables that follow reference will be made to " bathers " and " non-bathers ". " Bathers " denote men who either use or have used (if only very rarely) the baths at their present place of work, and " non-bathers " men who have never used the baths at their present place of work.

The prevalence rate for " bathers " was 31% (364 out of 1,153), and for " non-bathers " 8% (74 out of 948). The cultural results for the former were: *T. mentagrophytes* 175, *T. rubrum* 129, *E. floccosum* 7, *C. albicans* 1, multiple infections 20, and for the latter *T. mentagrophytes* 49, *T. rubrum* 19, *E. floccosum* 4, *C. albicans* 1, multiple infections 0.

To see whether frequency of bathing was important, " bathers " were divided into three groups, namely, daily " bathers ", weekly " bathers ", and others. The prevalence rates were 33%, 35%, and 21%. The last group (approximately 12% of the total number of " bathers ") was a composite one; it contained men who bathed as frequently as once a month and others who had not bathed for some years.

Nearly half the men examined gave a history of previous exposure. The prevalence rates were " bathers " with previous exposure 29% and without it 34%, " non-bathers " with previous exposure 13% and without it 3%. In Table 2 both " bathers " and " non-bathers " have been further subdivided according to whether they received their previous exposure in industry or the Forces or both. Previous exposure made little difference to the rates or species incidence for " bathers " but appeared very important for " non-bathers ". As might be expected, of the " non-bathers " those with a history of double exposure had the highest rate, and then came men who had used other industrial baths; those who had been in the Forces gave a lower figure—perhaps because many men had served in the war of 1914-18 only—and last came men with no exposure in either the Forces or industry (Table 2).

TABLE 2

EFFECT OF PREVIOUS EXPOSURE

Group	Men with Service in the Forces		Men with No Service in the Forces	
	Number	% Positive	Number	% Positive
Bathers: Men who have used other industrial baths	73	30	171	30
Men who have not used other industrial baths	298	29	611	34
Non-bathers: Men who have used other industrial baths	52	23	170	17
Men who have not used other industrial baths	231	10	495	3

The rates for the individual Services are given in Table 3; although the rates for ex-Naval men are the highest, the effect of Army service is more important since 80% of the ex-servicemen had been soldiers. Fraser (1939) noticed that sailors who had served abroad had higher rates than those who had stayed in home waters. We were unable to confirm this since only five of our ex-sailors had no foreign service, but our results for ex-army personnel show a similar trend. All who were non-bathers were divided into three groups: Those with service in the United Kingdom only (56 in all), those who had also served in Europe but not elsewhere overseas (91 in all), and those who had travelled farther afield (83 in all). The rates were 3·6%, 5·5%, and 18% respectively.

TABLE 3

EFFECT OF SERVICE IN THE FORCES

		Number	% Positive
" Bathers "	Royal Navy (including Allied Navies)	39	31
	Army	288	28
	Royal Air Force	33	36
	Others (including Merchant Navy)	11	36
"Non-bathers"	Royal Navy (including Allied Navies)	32	34
	Army	230	10
	Royal Air Force	16	6
	Others (including Merchant Navy)	5	20

Fourteen infected men gave no history of exposure in industry or in the Forces, but on special enquiry four of them said they used swimming baths. This leaves 10 men for whom the origin of infection remains obscure.

Correlation of Clinical and Laboratory Findings.—So far we have measured the effect of environmental factors by studying the laboratory results. The clinical findings are also of interest. The proportion of men with abnormal feet were as follows: " Bathers " with previous exposure 94%, and without it 94%; " non-bathers " with previous exposure 89%, and without it 84%. These four clinical rates are all much higher than the corresponding laboratory rates (see above). The correlation of the clinical and laboratory findings has been discussed (Holmes and Gentles, 1956), but it is important to note that whilst the clinical rates for all four groups were quantitatively similar, there were important qualitative differences between them. Thus the percentage of men with vesicular lesions increased from 2% for men with no history of exposure to 18% for " bathers ".

Variation from Place to Place.—For " bathers " the prevalence rates varied from place to place and so did the cultural results (Table 4). The highest rate, 53%, was at C and the lowest, 16%, at B where the baths had only been open six months. The commonest species of dermatophyte was usually *T. mentagrophytes*; but at Pit C *T. rubrum* was far the commoner and at Pit J the incidence of the two species was almost equal. Over half the multiple infections occurred at these two pits; at J multiple infections formed 12% of the total positives. Pit H was unusual in that no case of *T. rubrum* infection was detected there.

The exact reason why the rate at Pit C was so high is not clear. Possibilities considered were the number of men employed, the age of the baths, the composition of the bath-house floor, the method of cleaning; it was decided that the most likely reason was the presence of *T. rubrum* as infections with this species are difficult to treat.

TABLE 4

PREVALENCE RATES AND CULTURAL RESULTS FOR BATHERS

Place	Total No. of Bathers	% Positive	Microscopically Only	*T. mentagrophytes*	*T. rubrum*	*E. floccosum*	Two Species
B	—	—	—	—	—	—	—
C	206	53	10	13	81	—	5
D	135	36	6	37	5	—	—
E	129	33	6	30	6	1	3
F	62	18	—	8	3	—	—
G	71	19	2	9	1	1	—
H	141	22	3*	25	—	2	1
I	—	—	—	—	—	—	—
J	190	30	5	22	24	—	7
K	—	—	—	—	—	—	—
L	6	33	—	1	1	—	—
M	24	17	—	1	2	1	—
N	52	27	—	10	2	1	1
O	42	31	—	9	2	1	1
B[1]	95	16	1	10	2	—	2
Total	1,153	31	33	175	129	7	20

*Indicates one *C. albicans* (culture also obtained).

TABLE 5

EFFECT OF PREVIOUS EXPOSURE ON CULTURAL RESULTS FOR BATHERS

Place	Men without History of Previous Exposure			Men with History of Previous Exposure		
	T. mentagrophytes	*T. rubrum*	Total No. Examined	*T. mentagrophytes*	*T. rubrum*	Total No. Examined
Pit C	9	63	154	4	18	52
Pit J	12	13	105	10	11	85
All others	68	9	352	72	15	405
Total ..	89	85	611	86	44	542

Two most important species only, excluding multiple infections.

The effect of previous bathing is shown in Table 5. There is no evidence that the epidemics of *T. rubrum* are due to differences in the men's past bathing history.

For " non-bathers " the variation in the species incidence and the cultural results are given in Table 6. It was found that much, if not all, of the variation in the prevalence rates depended on the opportunities for infection in industry; the effect of service in the Forces was more evenly distributed. Figures obtained at Pits L and B will be used to illustrate this point, for, excluding small groups, these pits had the highest and the lowest rates respectively. L was a pit situated in a town and it was near baths built as long ago as 1913. Of the 142 " non-bathers " examined at this pit, 52 gave a history of using pit-head baths previously and 14 of them were infected. B was a pit in the country and it had no neighbouring baths before 1949. Only 15 men out of 200 had used pit-head baths and none were infected. Further details are given in Table 7.

TABLE 6

PREVALENCE RATES AND CULTURAL RESULTS FOR NON-BATHERS

Place	Total No. of Non-bathers	% Positive	Microscopically Positive	*T. mentagrophytes*	*T. rubrum*	*E. floccosum*	Two Species
B	200	3·5	—	5	1	1	—
C	11	9	1	—	—	—	—
D	14	—	—	—	—	—	—
E	24	4	—	1	—	—	—
F	10	10	—	1	—	—	—
G	5	—	—	—	—	—	—
H	7	—	—	—	—	—	—
I	146	6	1*	6	1	1	—
J	31	10	—	3	—	—	—
K	169	8	—	12	2	—	—
L	142	15	—	11	10	—	—
M	100	11	—	6	3	2	—
N	16	19	—	2	1	—	—
O	20	15	—	2	1	—	—
B[1]	53	—	—	—	—	—	—
Total	948	8	2	49	19	4	—

*Indicates one *C. albicans* (culture obtained).

TABLE 7

FURTHER EVIDENCE OF EFFECT OF PREVIOUS EXPOSURE ON RATES FOR NON-BATHERS

Laboratory Report	Men with Double Exposure in Industry and Forces		Men Previously Exposed in Industry Alone		Men Previously Exposed in Forces Alone		Men with no Known Exposure	
	Positive	Negative	Positive	Negative	Positive	Negative	Positive	Negative
Pit L	6	12	8	26	2	29	5**	54
Pit B	0	6	0	9	6	59	1	120

*One infected man known to use swimming baths.

As for " bathers" the commonest species was *T. mentagrophytes*. Forty-nine men were infected with this species; 40 of them had a history of exposure (two as swimmers). Only four men were infected with *E. floccosum*; three gave a history of exposure. There were no multiple infections. All the 19 men who were infected with *T. rubrum* gave a history of exposure (two as swimmers). It was noted that 10 men with this species came from Pit L, where they formed 48% of the total number infected; elsewhere the proportion was 17%. There were no cases of *T. rubrum* infection from the 42 " non-bathers " at C and J, the two pits where this species was common in " bathers ". We suggest that the high incidence at L is due in part to *T. rubrum* being endemic at a neighbouring colliery or collieries for eight out of the 10 men had used pit-head baths.

Method of Spread of Infection.—Gray (1911) and Sanderson and Sloper (1953) have referred to cross-infection via infected clothing. From general inquiries we believe that few of the men in our survey shared clothing, and if this is true then clothing cannot be important for our population. The main criticism of the other theory that infection is spread via the floors is the failure of the vast majority of attempts to isolate dermatophytes from them. Peck, Botvinick, and Schwartz (1944) made over 60 attempts and concluded from their negative results that either the fungi were not widely disseminated on floors, or they were washed down the drains too quickly to make isolation possible by the procedure used. So far as we are aware there are only two detailed reports of isolation of dermatophytes from floors. Adamson and Annan (1949) isolated *T. mentagrophytes* from a piece of skin found on the floor of a bath-house, and Ajello and Getz (1954) isolated the same species from a shower floor of a penitentiary in the U.S.A. Using a direct sampling method* we have been successful in isolating *T. mentagrophytes* from the floors of showers of three pit-head baths, the dressing-room of an athletic ground, and a public swimming bath. *T. rubrum*, which was the fungus present in over 80% of positive cases at Pit C, was isolated from the showers there.

*The essential of the method is the use of a velvet pad on a wooden stamp. Full details are given in a separate publication (Gentles, 1956).

Source of Infection in a New Bath-house.—The original sources of infection are multiple. After Dunkirk many baths, including C, were used by army units; nowadays infection is probably introduced mainly by ex-servicemen as individuals and by men from other pit-head baths. On the return visit to B it was found that men with a history of previous exposure were the first to use the baths, the others being more cautious. Moreover, it so happened that some men were included in both samples and all those originally proved to be infected were bathing. " Non-bathers " at this visit formed the only large group free of infection which also suggests that there had been some selection.

Seasonal Variation

Sanderson and Sloper (1953) have discussed seasonal variation under tropical conditions.

One pit with baths (C) was visited on three occasions, in March, June, and December, 1953. The infection rates for " bathers " were 50, 55, and 55% respectively. No one pit without baths was visited twice, but the visit to B was in February, to M in June, and to I in November. The return visit to B took place in 1954. No evidence of seasonal variation was detected; however, it should be mentioned that the predominant species at Pit C was *T. rubrum* and seasonal variation was not studied at a pit with baths when the predominant species was *T. mentagrophytes*.

Age

Men were divided into six age groups (Table 8). The rate for the lowest age-group (15-24) was 15% and for the highest age-group (65+) 17%; there was a peak rate of 25% at ages 35-44. When the men were divided up according to their bathing habits it was found that there was no real pattern to the rates for " non-bathers ", whilst that for " bathers " reached a flat level at age 45 and did not fall as the men grew older. The apparent fall with increasing age seen in the crude rates was due to the high number of " non-bathers " without exposure in the older age group.

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THE EFFECT OF AGE

Age in Years	Total		All Bathers		Non-bathers with Previous Exposure		Non-bathers without Previous Exposure	
	Number	% Positive	Number	% Positive	Number	% Positive	Number	% Positive
15–24	451	15	194	25	113	12	144***	4
25–34	419	22	261	28	109	15	49	4
35–44	389	25	244	32	74	19	71*	6
45–54	521	23	306	36	67	12	148	1
55–64	262	22	121	38	84	10	57	2
65+	59	17	27	37	6	0	26	0

*One infected man known to use swimming baths.

Duration of Present Employment

Men were divided into six groups. As with age there was a peak rate for the middle groups, but it was less marked. Allowances were made for bathing; once again there was no pattern for " non-bathers " whilst that for " bathers " reached a flat level at the six- to ten-year period. (Further details are given in the report to the M.R.C. Committee.)

On more detailed analysis it was found that for bathers with under one year's service and without previous exposure the rate was 7%, but for a similar group with previous exposure the rate was 23%. Admittedly, duration of employment is only a rough measure of the time over which the men had used baths, but this apparent lag in the increase of infection has been noticed by other workers (Vanbreuseghem, Peeters, and Tritsmans, 1952). It is consistent with the experimental work of Baer, Rosenthal, Rogachefsky, and Litt (1955) who found it difficult to infect human volunteers. However, this lag is not universal for there was a notable increase in the prevalence rate at Pit B within six months of the installation of baths. At the first visit the rate was 3·5%, and at the second visit it was 10%, or 16% (15 of 95) for " bathers " and 0% (0 of 53) for " non-bathers ". Even when allowances are made for the tendency at this colliery for men with a previous history of exposure to use the baths and for men without such a history to refrain, the rise is still appreciable—probably not less than 10%—from 6 to 16%.

Factors Peculiar to Mineworkers

Site of Work.—Carrié and Zantopf (1941) report a higher rate for underground workers than for surface workers. No comparison can be drawn as the bathing habits of the men were not given, nor were any details given of the conditions underground. Our rate for underground workers and for surface workers who had worked underground was 23% (369 out of 1,588), while for surface workers who had never been underground it was only 12% (22 out of 214). Much, but not all, of this difference could be attributed to their bathing history (Table 9) and to the fact that surface workers who had never been underground were often new entrants to the industry. Perhaps the feet of underground workers are more subject to minor trauma, which Kligman (1952) has shown to be important; or perhaps the surface worker who has never been underground may be more cautious and take greater care of his feet.

Nature of Work.—The number of surface workers was too small to be subdivided. The nature of employment underground appeared to be unimportant. (Details are given in the report to the M.R.C. Committee.)

Temperature Underground.—Three pits with baths (D, E, and J) had one seam distinctly hotter than the others. The hottest seam was at J, where a reading of 75° F. (wet bulb) was obtained with a whirling hygrometer. It appeared unlikely that the temperature affected the rates very much: however, the number of men involved was rather small.

The effect of working in a hot seam on the rates for " non-bathers " was to have been studied at Pit L. Unfortunately the hot seam was closed by the time we carried out our investigation.

Few pits in Great Britain have temperatures much higher than 75° F. (wet bulb) and when they do, the

TABLE 9
EFFECT OF SITE OF WORK

Site of Work	Bathers		" Non-bathers " with Exposure		" Non-bathers " without Exposure	
	Total	% Positive	Total	% Positive	Total	% Positive
(1) Underground	889	32	229	14	256	4
(2) Surface previously underground ..	77	34	51	12	65	1
(1) + (2) + others	975	33	282	14	331	4
Surface never worked underground ..	84	24	44	5	86	0

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TABLE 10
EFFECT OF WORKING IN HOT SEAMS ON REGULAR BATHERS

Place	All Regular Bathers		Surface Workers		Workers in Hot Seams		Workers in Other Seams	
	Total	% Positive	Total	% Positive	Total	% Positive	Total	% Positive
Pit J	151	34	27	33	57	37	62	34
Pit D	121	35	13	46	18	44	71	28
Pit E	97	43	8	50	26	42	50	40

maximum temperature is usually confined to one district or even part of a district (McLintock, 1954).

Inconvenience and Disability

No men examined by us were off work primarily for a skin complaint, but 2% gave a history of having lost time due to conditions diagnosed as foot ringworm by their own doctors. At two pits several men had stopped using the baths because they had had sore feet or else because they did not wish to become infected.

Apart from this, few men would state that they knew anything was wrong with their feet, possibly because to have done so would have been to admit that they should have been receiving treatment. Only 36 men were having treatment at the time they were examined; 16 of them were found to be infected. This is important because treatment might have led to the masking of infection and our failure to isolate fungus from clinically suspicious lesions.

Follow-up Examination

Pit C was visited in March, June, and December of 1953. We returned in November, 1954, to re-examine 100 of the regular bathers. The results for 80% of the men remained unchanged, 46 being infected both times and 34 being apparently free from infection both times. Ten men were positive the first time only and nine men were positive the second time only. One man could not be seen the second time. This makes a total of 65 infected at one time or another, 55 of them with *T. rubrum*.

From a study of the clinical findings it appeared (1) that some of the men found to be infected once only were really infected both times, (2) that some men had got better whilst others had become infected during this period. Four out of the five men who had got better were originally suffering from an infection with *T. mentagrophytes* and only one from an infection with *T. rubrum*.

Discussion

Our survey has dealt with specific groups of industrial workers in Great Britain but we believe the results are capable of more general application and therefore the discussion will not be confined to the problems of ringworm in these groups.

In most published reports on the incidence of foot ringworm there is a discrepancy between the number of men with clinically abnormal feet and the number proved to be infected on laboratory examination. Early workers, *e.g.*, Morris (1911), believed that this was because our laboratory methods were insufficiently sensitive and that if better techniques were available proof of infection would be obtained in the majority of such cases. For a time, from 1927 till 1936, when the finding of the mosaic fungus (Weidman, 1927) was accepted as evidence of infection, they appeared to be right. However, it is now generally agreed that mosaic is an artefact (Goldsmith and Hellier, 1954). The question of almost universal infection is therefore still being debated. Whilst some workers, *e.g.*, Linn and Magarey (1941), consider that most cases of ringworm can be diagnosed readily by methods such as we have used, others consider there are many minimal infections that are missed. It should be noted in this connexion that whenever possible we re-examined surplus material from men who had suspicious lesions but who were not proved to be infected; only one extra positive case was proved in this way. (Report to the M.R.C. Committee.)

Sulzberger and Baer (1955) believe that foot ringworm is not transmissible in the ordinary sense. They argue that dermatophytes are ubiquitous, that we are all infected from time to time, and that whether or not we suffer from foot ringworm depends on personal immunity. We agree that personal immunity is important but we disagree over the other points. There is no evidence that dermatophytes are ubiquitous in sufficient quantity to cause infection except perhaps in certain specialized places such as the floors of communal bath-houses. There is, moreover, good evidence that cross infection not only occurs but is the most important factor. Our reasons for holding this belief are as follows:—

Evidence from the Study of Non-bathers.—(*a*) Our low rates are similar to those of other workers (Phillips, 1944; Walker, 1950; Amrein, 1953). (*b*) It is implicit in the theory of universal infection that the effect of previous exposure should be short lived. This was not our experience. (*c*) Many of our "non-bathers" bathed daily at home; it is

difficult to see why only communal bathing should lead to a breakdown in immunity.

Evidence from the Study of Bathers.—(*a*) The variation in the prevalence rates and in the species distribution, in particular the high rate of *T. rubrum* at Pit C and its absence at Pit H, are greater than one would expect if exposure were unimportant and the effect of bathing were non-specific. It was the absence of a noticeable difference in the species incidence that led Hopkins *et al.* (1947) to support Sulzberger's views. (*b*) Dermatophytes were cultured from the floors of four bath-houses and on each occasion the species cultured corresponded to that found on the majority of men using the bath-houses. (*c*) It may apparently take months of regular daily bathing to cause infection, *cf.*, Vanbreuseghem *et al.* (1952) and the experimental work of Baer *et al.* (1955). This suggests that both heavy exposure and a breakdown in personal immunity are necessary at one and the same time. (*d*) The absence of seasonal variation and the results of the follow-up examination at Pit C suggest that once a man is infected either the infection remains detectable or else he rids himself of infection completely.

Multiple Infections.—No multiple infections were detected in 74 infected " non-bathers " but there were 20 cases among the 364 infected " bathers ". The highest incidence of these infections (13·7% of infected bathers) was at Pit J where there was an almost equal number of men infected with one or other of the major species.

Summary and Conclusions

An epidemiological survey of foot ringworm was conducted in different parts of Great Britain and in communities some of which did and some of which did not have communal baths. Most, but not all, of the men examined worked in the coal-mining industry. Comparisons of the clinical findings and mycological infection rates between the various groups of men suggested that, although previous exposure and working conditions were related to the prevalence of infection, the habitual use of communal baths was the most important factor. The distribution of the dermatophyte species varied from place to place, and on four occasions when a dermatophyte was isolated from the floor of a bath-house the species corresponded to that occurring most commonly in the men who were using it. These findings are discussed in relation to the hypothesis concerning the spread of foot ringworm.

It is concluded (1) that fungus infections of the feet are not universal; (2) that a specific exposure and a breakdown in personal immunity are necessary for infection to occur; (3) that in Great Britain these conditions occur together most commonly in those who use communal baths; (4) that in this country, other factors such as age, seasonal variation, and conditions of employment are of minor importance.

We wish to thank the medical services, the managements, and the men of the National Coal Board and the British Electricity Authority for their cooperation.

Sincere thanks are also expressed to the staff of the department of Medical Statistics and Epidemiology, the London School of Hygiene and Tropical Medicine; the University of Glasgow and Professor John Walton, who provided laboratory accommodation; the University of Leeds for library facilities; Miss C. O. Dawson, Department of Bacteriology, the University of Glasgow; the medical artist of the Department of Pathology, the Welsh National School of Medicine; and the many colleagues who have discussed our problem with us.

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BY

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The contact of quartz with lung tissue and the ensuing inflammatory reaction is the first and most important phase in the development of silicosis. The organism can to some measure influence this reaction by its own neurohumoral regulation, as not everybody falls prey to the disease, and if he does, not to the same degree even under similar exposure. On the other hand, a chronic course of silicosis changes the neurohumoral position of the whole organism (Vyskočil, 1954).

In recent years clinical therapy has been enriched by powerful hormones, especially A.C.T.H. and cortisone, which can inhibit the formation of fibrotic tissue in inflammatory processes. Silicotics clearly do not suffer from any pathological changes of the endocrine glands, even when exhaustion by chronic illness is evident (Vyskočil, 1954), but it has been thought interesting to try and effect a decrease in the fibrosis of the silicotic lung by the administration of therapeutic, not physiological, doses of A.C.T.H. and cortisone. Many authors have used these substances in animal experiments and in man. The results show that a formed silicotic nodule can hardly be influenced by either cortisone or A.C.T.H. (Schiller, 1953; Curran, 1952; Ducommun and Ducommun, 1951; Margarey and Gough, 1952; King, Harrison, and Attygalle, 1955). A more pronounced effect was observed when cortisone or A.C.T.H. was being given from the beginning of the administration of quartz. Cortisone or A.C.T.H. retards the transport of dust to the phagocytes from lung tissue into hilar nodes (Stacy and King, 1954); they enhance quartz dispersion in the whole organism (Policard and Tuchmann-Duplessis, 1951, 1952) which is unfavourable, but on the other hand they retard the growth of the silicotic nodule and the change of reticulin fibres into collagen (Stacy and King, 1954; Ducommun and Ducommun, 1951; Harrison, King, Dale, and Sichel, 1952; Margarey and Gough, 1952; Marenghi and Rota, 1953). The doses were usually large (Michalová, 1954). All these experiments are of more value in understanding pathogenesis than in the clinical therapy of silicosis. The administration of A.C.T.H. and cortisone would have to be prolonged and inhibition of various endocrine organs would be encountered.

We have tried the prolonged administration of very small doses of A.C.T.H. alone or in combination with insulin, which in some respects is an antagonist and in others a synergist of A.C.T.H. Insulin can provoke endogenous A.C.T.H. formation by hypoglycaemia (Bartelheimer and Cabeza, 1942; Knick, 1954) which could be useful as an aid against the possible hypophyseal atrophy in prolonged exogenous A.C.T.H. administration.

Schiller (1953) proved by experiment that depot insulin injected in doses of 0·004 i.u. into white mice can retard fibrotic tissue formation after intraperitoneal quartz administration (10 mg.) in the first 30 days.

It remained to find out how to administer insulin, whether together with A.C.T.H. or on alternate days. To solve this problem we divided the animals into several groups: one group was injected with A.C.T.H. alone, another with A.C.T.H. and insulin, a third one with A.C.T.H. and insulin on alternate days, and a fourth with insulin alone.

One group of animals received salicylates, as we wanted to determine the relation of A.C.T.H. and salicylates with regard to their effects upon the inflammatory reaction and upon the endocrine organs. The role of salicylates in silicosis has been discussed in another paper (Vyskočil, 1955).

We have found quartz injections into the anterior eye chamber in rabbits a convenient method for following the development of silicotic granuloma. Several interesting conclusions with regard to the pathogenesis of silicosis can be drawn in this manner, the main advantage being that the whole development of the granuloma can be viewed under the transparent cornea and the necessary histological sections can be taken at various intervals.

Methods and Experimental Animals

The injections of quartz dust into the anterior eye chamber were carried out under strictly aseptic conditions. Two days before the injection penicillin drops were put into the conjunctival sac. Granulomas were produced by the injection of 0·05 c.cm. of a 5% quartz dust suspension; the quartz particles were all under 10μ, most being between 2 and 3μ. The cornea and conjunctival sac were anaesthetized with 4% novocaine in drop form.

The bulbus was fixed by grasping the bulbar conjunctiva near the upper external end of the corneal limbus and injecting against this light pull about 1 mm. from the limbus through the cornea and into the anterior chamber. Corneal fluid was not withdrawn; the dust was injected immediately after suspension in a tuberculin syringe. After enucleating the eye the upper bulbar pole was marked so that the vertical meridian could be observed when cutting the histological sections; this is important because the dust particles sink to the floor of the anterior chamber.

We used 50 rabbits of similar weights, all females. They were divided into six groups. The experiment lasted six months, and therapy was started on the first day. Group I contained nine untreated controls. Group II (10 rabbits) received daily for the first two and a half months 0·1 mg./kg. A.C.T.H. and then for three and a half months 0·2 mg./kg. subcutaneously. Group III (six rabbits) was injected with 0·25 i.u./kg. insulin daily subcutaneously. Group IV (six rabbits) received A.C.T.H. (same quantity as group II) and 0·25 i.u./kg. insulin on alternate days. Group V (10 rabbits) received A.C.T.H. (0·1 to 0·25 mg./kg.) and insulin (0·25 i.u./kg.) daily together. Group VI (nine rabbits) received for the first two and a half months 0·250 g./kg. sodium salicylate in a 10% solution by tube or 0·250 g./kg. salicylamide in food; for the remaining three and a half months the dose was lowered to 0·125 g./kg.

These doses of both hormones and salicylates were effective as we observed two- to five-fold increases in circulating pseudo-eosinophils within two hours. Insulin (0·25 i.u./kg.) caused an average decrease of fasting blood sugar of 45% within an hour.

During the first month the inflammatory reaction was observed almost daily, then at monthly intervals. In macroscopic examination we took account of the dilated vessels and their attachment to the granuloma; the turbidity of the chamber liquid, the size of whitish film adhesion above the nodule; the degree of pigmentation round them and the size and deformity of the pupil. By these criteria the inflammatory reaction was classified between 0 and 6 points. All animals were killed after six months.

Histological sections were stained with haematoxylin and eosin, the adrenals with Sudan III for the estimation of lipids, and the hypophysis by the Mallory method for the estimation of chromophobe, eosinophil and basophil cells. All endocrine glands were weighed immediately upon extraction.

Besides these 50 animals in the six groups designed to follow the effect of hormones and salicylates upon the development of the silicotic granuloma, we had in the experiment rabbits into which were injected non-fibrogenic dusts—brick (five rabbits) and scoria (seven rabbits) into one eye and quartz dust into the other eye. Another group of 20 animals was inoculated with quartz dust and left untreated; these animals were killed at various intervals from a fortnight to six months.

Results

Macroscopic examination one day after the quartz injection showed considerable white turbidity in the whole eye chamber and little clouds of white fibrin. This lasted for about three days, then the turbidity began to clear. Round the whole limbus a slight ciliar injection could be seen. As the turbidity decreased, films of fibrin above the quartz deposits could be seen. The relatively calm phase of the reaction occurred about the tenth day, but even then the whitish film above the injected quartz in the iris persisted. Contrasting with this is the fact that the fibrin formed above non-fibrogenic dusts (brick and scoria) was dissolved during these days and finally vanished.

About the tenth day pigment began to migrate around the dust deposits. Anterior synechiae and pupillar deformity sometimes occurred. In the third to fifth weeks after the quartz injection the inflammatory reaction increased, this time without any significant white turbidity in the chamber fluid. Instead we observed dilatation of the iris vessels, increased conjunctival injection, and sometimes vascular dilatation in the posterior corneal wall as well. This increased reaction calmed down within a fortnight with the exception of vascular dilatation which persisted thereafter.

Histological examination of the eyes on the 17th to 30th day revealed nodules of increased histiocytes and sometimes slightly pink, structureless material in the iris and irido-corneal angle. The nodules contained relatively large histiocytes with large vacuolized centres of evidently phagocytic nature.

Conglomerations of pseudo-eosinophil cells, lymphocytes, and plasma cells were often found. Fibrin films were in some cases encountered above the nodules, but lamellar fibrosis was never seen (Fig. 1).

Histological examination after four to six months revealed granulomas in the oedematous iris tissue, in the irido-corneal angle, and in the corpus ciliare. The main granuloma constituents were histiocytes, fibroblasts, lymphocytes, pseudo-eosinophils, histiocytes with phagocytosed pigment liberated from the migrating pigment epithelia, and extracellular pigment; sometimes also disintegrated sclera. This phase was marked by the finding of massive lamellar fibrosis. We want to emphasize that these firm, slightly cellular fibrotic lamellae composed of collagen, eventually of hyalin (equal to third to fifth grade by the Belt and King classification, 1945), were only formed above the nodules in the free irido-corneal angle lying against the posterior corneal wall, and never on the opposite part of the nodule in the iris stroma or the irido-corneal junction or corpus ciliare which contain a rather dense fibroblastic net even under normal conditions (Figs. 2 and 3). What we see here is evidently mostly fibrotic organization of plasma exudate.

The group which was injected with non-fibrogenic dusts also had whitish turbidity and fibrotic films in the first days after the inoculation, but these were smaller and vanished more quickly, within the first six days. The vessel reaction in the following three to five weeks, found with quartz dust, did not appear. Also the deposits contained a smaller amount of pigment. Histological examination did not reveal any fibrotic lamellae above the nodule in the free irido-corneal angle.

Group I: Quartz Alone (Control).—The control group of nine untreated animals with which other groups (II—VI) were compared had the greatest amount of whitish turbidity in the anterior eye chamber in the first three days. This was classified as 3·2 points. The reaction slowed down until on the tenth to seventeenth day it began to increase again, this time mostly by vascular dilatation. There was no secondary infection. After six months the macroscopic reaction was classified as 2·7 points.

Histological examination revealed silicotic granulomas as described above in the iris stroma, corpus ciliare, and the irido-corneal angle. Fibrotic lamellae, slightly cellular and almost collagenous up to completely acellular in the free irido-corneal angle, were found in 66% of the animals (Figs. 4 and 5).

The average weight increase was 15 dkg. (the animals ate less at the beginning because of the trauma). Average weights of the endocrine glands are to be found in Table I.

Group II: Quartz and Daily A.C.T.H. Administration.—Macroscopic reaction was smaller in the first days than in the control group (2·5 points). Two animals showed secondary infection in the eye chamber and conjunctiva during the first month which is understandable with A.C.T.H. administration. After six months the silicotic granuloma in animals with no infection was classified as 1 point.

Histological examination revealed silicotic granulomas with identical cellular elements as in the control group, but the granulomas seemed to be somewhat smaller and contained less pigment in histiocytes and extracellularly. In two animals (25%) only could fibrosis in the corneal angle be found (Fig. 6a). One animal had several layers of fibroblasts localized between the histiocyte infiltrates in the iris and the Descement membrane. There was neither collagen nor hyalinization (Fig. 6).

The average weight increase was 23 dkg. The adrenals registered a statistically significant weight increase when compared with that of the control group ($P < 0.01$). Histological examination revealed a slightly enlarged adrenal cortex, mainly in the zona glomerulosa and the zona fasciculata, and an enlarged adrenal medulla with dilated sinusoids.

TABLE I

EXPERIMENTAL RESULTS

Group	Number of Animals	Average Weight (kg.)		Incidence of Secondary Infection (%)	†Macroscopic Classification		Incidence of Lamellar Fibrosis (%)	Glucose Fall (%) after 0·25 u./kg. Insulin after 6 Months	Average Weight			
		Before Experiment	After 6 Months		First 3 Days	After 6 Months			Adrenals (g.)	Hypophysis (mg.)	Ovary (g.)	Uterus (g.)
I	9	2·30	2·45	0	3·2	2·7	66	45	0·41	20·04	0·37	4·07
II	10	2·15	2·38	20	2·5	1·0	25	35	0·54*	24·30	0·40	5·16
III	6	2·24	2·40	17	3·2	2·2	66	29	0·41	27·4	0·21	3·10
IV	6	2·37	2·41	17	2·3	1·2	50	37	0·45	29·9**	0·39	4·94
V	10	2·21	2·70	10	3·0	1·8	30	29	0·52	21·02	0·52	8·52*
VI	9	2·45	2·41	22	2·6	1·3	0	26	0·51***	20·20	0·47	5·46

Statistical evaluation with regard to the control group gave* = $P < 0.01$; ** = $P < 0.02$; *** = $P < 0.05$.
† Excluding animals with secondary infection.

FIG. 1.—Silicotic granuloma in the anterior eye chamber in rabbit after 18 days. Stained with haematoxylin and eosin. × 240.

FIG. 2.—Silicotic granuloma in the anterior eye chamber in rabbit after six months. From the left: iris with incorporated quartz dust and liberated pigment; fibrotic lamellae; cornea. Stained with haematoxylin and eosin. × 240.

FIG. 3.—Massive lamellar fibrotic composition stained after Gömöri (iron-silver). In the right upper corner white stripe—the Descement membrane. × 240.

FIG. 4.—Silicotic granuloma in the anterior eye chamber in control animals after six months. Stained with haematoxylin and eosin. × 12.

FIG. 5.—Silicotic granuloma in the anterior eye chamber in control animals after six months. Stained with haematoxylin and eosin. × 12.

Group III: Quartz and Daily Insulin Administration.—The inflammatory reaction in these animals was not very much different from that in the control group during the first three days and received 3·2 points. Further development was also similar to that in the control group. Secondary infection of the eye chamber was found in 17% of the animals. After six months the reaction by macroscopic examination was classified as 2·2 points.

Histological examination revealed the same cellular composition of silicotic granulomas as in the control group. Sixty-six per cent had a massive fibrotic reaction in the form of slightly cellular parallel fibrotic lamellae. The granulomas contained more pigment (Fig. 8) than in the control group. This group was the weakest of all. Weight increase was only 16 dkg., though in the first two months it

FIG. 6.—Silicotic granuloma in the anterior eye chamber in animals given A.C.T.H. after six months. Stained with haematoxylin and eosin. × 12.

FIG. 6A.—Same as Fig. 6A. × 240.

FIG. 7.—Silicotic granuloma in the anterior eye chamber of rabbits given A.C.T.H. and insulin after six months. Stained with haematoxylin and eosin. × 12.

FIG. 8.—Silicotic granuloma in the anterior eye chamber in rabbits given insulin after six months. Stained with haematoxylin and eosin. × 12.

FIG. 6

FIG. 7

FIG 6A

FIG. 8

FIG. 4.—Silicotic granuloma in the anterior eye chamber in control animals after six months. Stained with haematoxylin and eosin. × 12.

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FIG. 6

FIG. 7

FIG 6A

FIG. 8

FIG .9.—Silicotic granuloma in the anterior eye chamber of rabbits given insulin and A.C.T.H. on alternate days after six months. Calcification of sclerotic lamellar tissue. Stained with haematoxylin and eosin. × 12.

appeared better. Of the endocrine glands, the hypophysis was enlarged by weight, the ovaries and uteri were decreased. Histological examination of these organs revealed dilated sinusoids in the adrenal medulla with unchanged size, few and atrophied islets of Langerhans in the pancreas, and decreased eosinophil cell content in the adenohypophysis.

Group IV: Quartz and A.C.T.H. and Insulin on Alternate Days.—The reaction during the first three days received 2·3 points. Secondary infection was observed in 17% of the animals. After six months the reaction was classified as 1·3 points.

Histological examination revealed similar granulomas to the control group. In 50% fibrotic lamellar structures were observed between the iris and cornea. One animal had commencing calcification in the lamellar fibrotic structure (Fig. 9).

The average weight increase was 4 dkg., and of the endocrine glands, that of the hypophysis was increased ($P < 0.02$). Histological examination revealed dilated sinusoids in the normal adrenal medulla and atrophied islets of Langerhans.

Group V: Daily Combined A.C.T.H. and Insulin Administration.—The inflammatory reaction in the first three days was not remarkably influenced, and a change for the better was only observed after the first months. Secondary infection was seen in 10% of the animals. Silicotic granuloma after six months was classified with 1·8 points.

Histological examination revealed granulomas of a composition similar to that in the control group, but fewer fibrotic lamellae in fewer animals could be found (30%). An example of a silicotic granuloma without massive fibrosis in the free irido-corneal angle can be seen in Fig. 7.

The average weight increase was 49 dkg., the largest of all, and of the endocrine glands, an increase in weight was found in the adrenals, and ovaries, and in the uterus. Histological changes consisted of an increased adrenal medulla with dilated sinusoids and slightly enlarged islets of Langerhans.

Group VI: Daily Salicylates.—The first reaction was marked with 2·6 points. It was smaller than in the control group. Secondary infection affected 22% of the animals. After six months the inflammatory reaction received 1·3 points.

Histological examination revealed granulomas with histiocytes, pseudo-eosinophil, and giant cells. The pigment content seemed to be smaller. Massive fibrotic lamellae could only be found in one case which also had secondary infection in the anterior eye chamber and so was not included in the final classification. One animal had in the posterior corneal surface only lamellar fibroblast layers without collagen fibres and hyalinization. Weight decrease averaged 4 dkg. (the gastric mucosa suffering particularly from the high salicylate dosage). Weighing the endocrine glands revealed a slight increase in the weight of the adrenal ($P < 0.05$). Histological examination revealed a constricted zona glomerulosa in the adrenal cortex (by Na salicylate), dilated sinusoids in a slightly increased adrenal medulla, and decreased eosinophilic cells in the adenohypophysis.

Conclusions

Our experiments show that quartz dust, like other dusts, produces in the tissues at first a normal inflammatory reaction, but it is capable of adsorbing strongly plasma, tissue, and cellular proteins on its

surface, on account of its physical properties there (Jäger, 1950; Bohun, 1954). Later on elution of silica seems to take place and its reaction with the surrounding substances.

The adsorbed proteins become denatured and provoke an increased delayed reaction which we observed in the anterior eye chamber during the third to fifth weeks. We saw massive fibrotic lamellae only in the free angle of the anterior eye chamber between the iris and the cornea, where there was exudation from dilated vessels and fibrin adhesion. Plasma proteins were invaded with cellular elements, especially macrophages, which later changed into fibroblasts.

Policard and Rollet (1931), who injected quartz into rabbits intracorneally, could not find the typical lamellar fibrosis round the deposits. The cornea contains few vessels and its firm consistency does not allow production of larger exudate. Our experience in histological examinations of quartz deposits in the cornea was similar: the dust which remained in the injection channel descended between the corneal layers to the lower limbus pole without forming the typical lamellar fibrotic structures and only a small chronic inflammatory infiltrate with histiocytes could be found.

In studies of fibrosis in the silicotic lung all authors insist upon the formation of fibrotic tissue in the interstitial free space. Our experiments show that in the lung, coniophage and liberated quartz conglomerations probably appear not only in the interstitial tissue but in the alveolar lumen as well, and that here the fibrotic exudate organizes. The lung alveolus with open spaces and a rich blood supply is therefore well adapted for the formation of massive fibrosis.

We found retarded fibrinolysis above the quartz deposits; this shows where the silicotic lung process might be similar to the tuberculous. In the tuberculous lung an increased antifibrinolysin level may be found (Rosenmann, 1937).

In our experiments, aimed at influencing the development of the silicotic granuloma, the results show that salicylates, A.C.T.H. alone and in combination with insulin, when administered simultaneously, have the most pronounced effect upon the growth of the silicotic granuloma. Both macroscopic and histological evaluations revealed quantitative changes, never qualitative, and the whole effect was only slight.

The A.C.T.H. dosage was low and did not cause any deterioration in the general state of health of the animals. Nor did we find any significant weight or histological changes in the endocrine organs, with the exception of the adrenal. Histological examination showed that salicylates in large doses were the most influential agent. When comparing the action of salicylates with that of A.C.T.H. administered in very small doses, we saw smaller enlargement of the adrenal and smaller histological changes so we suggest that salicylates have their own specific effect in the periphery. Salicylates stimulate the system: the central nervous system and the hypophysis but the adrenals only secondarily (Vyskočil, in the press).

Insulin had no favourable influence upon the silicotic granuloma. This is in contrast to the findings of Schiller (1953), but this author administered depot insulin and apparently the comparison was difficult because he used white mice, gave smaller amounts of insulin, and only followed the response to quartz in the peritoneal cavity for 30 days.

The assumption that endogenous A.C.T.H. production stimulated by insulin would have a favourable effect upon the fibrosis was not confirmed. The animals deteriorated after insulin, while the inflammatory reaction itself did not benefit, even in the first days. Insulin administered on alternate days with A.C.T.H. did not prove our assumption that it would stimulate the hypophysis to A.C.T.H. production and so prevent hypophyseal atrophy after the organism had been overloaded with exogenous A.C.T.H. This group of animals also deteriorated after insulin and the reaction to quartz was the same as in the control group. Insulin apparently had a greater effect than A.C.T.H. Both these groups of animals had slightly heavier hypophyses, dilated sinusoids in the adrenal medulla, and atrophied islets of Langerhans due to adaptation to insulin. All these changes were more pronounced in the insulin group than in the combined insulin-A.C.T.H. group and, besides, the insulin group had smaller ovaries and uteri and fewer eosinophil cells in the adenohypophysis.

In contrast to this, the group receiving A.C.T.H. and insulin daily together had a smaller fibrotic reaction than the control group and their general state of health was the best of all. All animals put on weight and had enlarged ovaries and uteri and even slightly enlarged islets of Langerhans.

The mutual relation of A.C.T.H. and insulin and questions connected with their therapeutic administration and histological findings in endocrine organs are discussed elsewhere (Vyskočil and Dufka, 1956).

In conclusion we may say that our experimental study has contributed more to our knowledge of the pathogenesis than to either the therapy or prophylaxis of silicosis. The results in animals do

not point towards anti-inflammatory hormone administration in man. The formation of fibrotic nodules could still be observed, though in a smaller number of animals. Though the group with combined A.C.T.H. and insulin administration had no large changes in endocrine function, the danger of disturbing this function still exists. But we should not overlook the favourable effects of salicylates on secondary inflammations in the silicotic lung (Vyskočil, 1955), as we may thus prevent the development of more massive fibrotic lesions. Our endeavours in research on clinical silicosis prevention must be directed toward prevention of inflammatory lung disease, especially tuberculosis.

Summary

Massive lamellar fibrosis after quartz inoculation was only produced in rabbits in the free space of the irido-corneal angle and mainly by organization of the plasma exudate.

The development of silicotic granuloma was reduced by salicylates, by A.C.T.H. alone, and by A.C.T.H. given with insulin and at the same time in that order.

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THE PREVALENCE OF TUBERCULOSIS AT NECROPSY IN PROGRESSIVE MASSIVE FIBROSIS OF COALWORKERS

BY

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The accumulation of dust particles in the lungs may produce harmful changes. At one time it was thought that serious damage to the lungs occurred only when the dust had a high content of free silica, but it is now accepted that coal, which contains only a small amount of free silica, can be associated with severe, and fatal, lung fibrosis (Gough, 1940).

It is well recognized that in classical silicosis, *i.e.*, the disease associated with a dust rich in free silica, tuberculosis is a frequent complication. The common type of pneumoconiosis seen in coalworkers has two forms: (*a*) a simple type without significant fibrosis and (*b*) a complicated type with severe fibrosis which is believed to be caused in most cases by tuberculosis in a lung already loaded with dust (Fletcher and Gough, 1950, Cochrane, Miall, Clarke, Jarman, Jonathan, and Moore, 1956). Strong pathological evidence of tuberculosis is present in many cases at necropsy. Some years ago a search was made in our respective departments for tubercle bacilli using routine cultural techniques and guinea-pig inoculation tests; in a series of 75 cases of massive fibrosis 25 (33%) were found positive (Fletcher, 1948). Other workers, whose results will be discussed later, did not make a systematic search for tubercle bacilli in all cases, and their conclusions were largely based on histological evidence. It therefore seemed possible that if a more vigorous search were made for tubercle bacilli, using improved methods of sampling the lung combined with the most efficient bacteriological methods, a larger proportion of positive cases might be found. The purpose of this paper is to report the findings of such an investigation.

Pathology of Coalworkers' Pneumoconiosis

All coal-miners have coal dust in their lungs, but many have no significant secondary pathological changes and suffer from no symptoms. At necropsy it is customary to recognize three main types of lesion.

Coal Nodules.—The lungs show foci of dust scattered almost uniformly throughout. Heppleston (1953) described the essential lesions as deposits of dust-laden macrophages enmeshed in a reticulin framework forming cuffs around the respiratory bronchioles. In some cases the foci contain and are surrounded by focal emphysema (Gough, 1940; Heppleston, 1953). Collagen fibres are scanty, and are mostly associated with older foci. The frequency and severity of focal emphysema increases with age.

Mixed Dust Nodules.—The lesion is a small fibrous nodule usually not more than 1·5 cm. in diameter and heavily impregnated with coal dust. Any such nodule under 3 cm. in diameter is included in this group. The nodule contains much collagen with fibres usually showing no well defined arrangement but which are sometimes disposed radially. The collagen often undergoes hyaline changes and there may be central ischaemic necrosis. The appearances of a nodule may be in some cases indistinguishable from an old healed tuberculoma containing coal dust. The lesions are termed "infective nodules" by those who consider that infection is a factor in their production. Others believe that the fibrosis is due to the silica in the dust and use the term "mixed dust nodules" (Di Biasi, 1954).

Progressive Massive Fibrosis (P.M.F.).—The lesion consists of a dense mass of collagen containing a large amount of coal dust. James (1954) accepted any lesion larger than 3 cm. in diameter as a lesion of massive fibrosis, and we have followed this definition. In almost all cases in this series the lesions were considerably larger than 3 cm. in

diameter. The collagen fibres show no definite arrangement, and the edge of the lesion is usually irregular. Most of the bronchi and vessels within the mass are obliterated, and the vessels at the periphery often show advanced endarteritis (Wells, 1954). Some patches of the collagen may be hyaline and often, presumably because of avascular necrosis, a cavity appears in the mass. The lesions are usually large and bilateral, the common sites being the posterior parts of the upper lobes. Massive fibrotic lesions are associated with simple pneumoconiosis in the rest of the lung. The site of the lesions, the endarteritis, and cavitation are similar to those in tuberculous infection.

In experiments with guinea-pigs Zaidi, Harrison, King, and Mitchison (1955) found that the combined action of coal-mine dust and tubercle bacilli of low virulence produced massive pulmonary fibrosis, whereas the dust alone or the bacilli alone did not produce any fibrosis. Serial cultures of the lungs yielded growth of tubercle bacilli up to, but not beyond, 120 days. It may be that a similar process occurs in man and accounts for the failure to isolate tubercle bacilli from the majority of massive fibrotic lesions. The radiological appearances of early massive fibrosis and tuberculosis are often sufficiently similar to make them indistinguishable (Cochrane, 1954), and in the Rhondda Fach survey it was found that the prevalence of tuberculosis in miners was less than among non-miners unless massive fibrosis was included as a form of tuberculosis (Cochrane, Cox, and Jarman, 1952).

In the investigation reported here necropsy material from coalworkers was examined macroscopically, histologically, and bacteriologically for evidence of tuberculosis. Special efforts were made to release tubercle bacilli from the fibrotic masses before examination by animal inoculation test and culture.

Choice of Material

A large number of cases of suspected pneumoconiosis in coalworkers come to necropsy in Wales. The disease is compensatable, and deaths from it are referred to the coroner; necropsies are performed at the request of the coroner.

In this study 98 cases of massive fibrosis formed the first group and a further 20 cases forming a second group were added later. Each group was made up of consecutive cases on which necropsy was performed by two of the authors (W.R.L.J. and D.R.). Included for comparison were 18 cases of mixed nodular pneumoconiosis and 17 cases of simple pneumoconiosis. All these cases formed part of the routine post-mortem material during the years 1952-54 inclusive.

Collection of Material

At necropsy a swab was taken of the main bronchus immediately after the lungs had been removed from the thorax. This swab was taken in order to establish what might have been the result of an examination of a specimen of sputum had such a specimen been taken shortly before death. It was known from previous experience that tubercle bacilli are found in only very few cases of massive fibrosis during life, and it was thought desirable to establish whether or not this state of affairs persisted until death, *i.e.*, whether the tubercle bacilli so often found in the fibrotic mass after death are entirely enclosed in tissue or are escaping in the sputum.

About 25 g. of the fibrotic mass was excised from the lung and put in a sterile jar along with any hilar lymph nodes draining the area.

The cases of mixed nodular pneumoconiosis and simple pneumoconiosis were similarly examined by taking a large piece of lung containing nodules or dust foci and the associated hilar glands.

Methods of Examination

Swab of bronchus.—The swab was immersed in 2·5% oxalic acid for 45 minutes, and was then rubbed over the surface of Lowenstein-Jensen's medium (two tubes).

Fibrous Mass.—The mass and hilar gland were placed in a metal Waring blender together with 20 ml. of nutrient broth. After about two minutes at 15,000 r.p.m. the tissue was macerated, but sometimes it was necessary to treat it for a second period. At the end of this treatment the tissue, though macerated and appearing to be a uniform mush, could not be drawn up into a syringe as collagen fibres blocked the needle. Grinding the mush with a pestle and mortar, even along with sand, had no advantages as the collagen became knotted into a firm mass. As the liquor was not thought to be the best sample obtainable the mush was treated with NaOH. Equal volumes (2 ml.) of 4% NaOH and mush were mixed and held at 37 C. for periods not exceeding 30 minutes. This produced considerable homogenization of the mush which was now diluted with 25 ml. of sterile distilled water and centrifuged at 3,000 r.p.m. for 30 minutes. The deposit was neutralized with 3% HCl, and cultures were made on Lowenstein-Jensen's medium and in Kirschner's medium (two tubes of each). At the same time a guinea-pig was inoculated. The whole of the deposit from 2 ml. of mush (representing approximately 1 g. of tissue) was used for these tests, roughly equal volumes being cultured on Lowenstein-Jensen's or in Kirschner's medium or inoculated into a guinea-pig. A study of a number of homogenizing agents by a working party of the Public Health Laboratory Service (1952) on the laboratory diagnosis of tuberculosis showed NaOH to give the highest success rate for culture of tubercle bacilli from sputum. These findings have recently been confirmed by Gray, Clarke, and Johnstone (1954) for *Mycobacterium tuberculosis* H37Rv.

Nevertheless, as an additional check in an extra 20 cases of massive fibrosis the macerated tissue, undigested with alkali, was injected into guinea-pigs protected with gas gangrene and tetanus antitoxins. This was thought

desirable in case NaOH in the presence of coal dust is harmful to tubercle bacilli. The material was not very satisfactory for injection, as little more than the liquor was drawn up into the syringe.

Histology.—In all cases two or more blocks of tissue from the lesions in each lung were taken for histological examination. Sections were stained by the haematoxylin and eosin method, and tuberculosis was diagnosed only if all the classical features were present.

Results

The cases were classified according to the type of pneumoconiosis and to the presence or absence of tuberculosis as judged by the naked-eye or histological changes. With a few exceptions the cases judged tuberculous showed both naked-eye evidence of the disease and the characteristic histological changes. The cases were classified before the results of the bacteriological examinations were known. The results are shown in Table 1,

TABLE 1

ISOLATION OF TUBERCLE BACILLI FROM CASES OF COALWORKERS' PNEUMOCONIOSIS

Pathological Classification	Bacteriological Result		
	Positive	Negative	Total
Type 1			
Simple pneumoconiosis (without tuberculosis)	2	15	17
Type 2			
Mixed nodular pneumoconiosis	0	16	16
Mixed nodules with tuberculosis	2	0	2
Type 3			
Progressive massive fibrosis	8	72	80
P.M.F. with tuberculosis	31	0	31
P.M.F. with doubtful tuberculosis	2	5	7

and, since the results for the 20 cases of massive fibrosis examined by injecting untreated macerated lesions into guinea-pigs did not differ from those of the first series, the two groups are considered together.

There were in all 118 cases of massive fibrosis and tubercle bacilli were isolated from 41 (35%). Thus refinement of techniques did not yield a greater proportion of successful isolations than had been obtained in earlier studies.

There was a very close agreement between the naked-eye and histological assessment and the bacteriological result; of 31 cases of massive fibrosis showing tuberculous tissue changes (naked-eye and microscopical), all were bacteriologically positive; of 80 cases of massive fibrosis with no obvious tuberculous changes, eight were positive; seven showed some appearances suspicious of tuberculosis and two were positive. All 16 cases of mixed nodular pneumoconiosis without tuberculous tissue changes were bacteriologically negative.

The finding of tubercle bacilli in two out of 17 cases of simple pneumoconiosis is surprising and may be the result of including the hilar glands in the material examined.

Discussion

Gooding (1946) reported post-mortem pathological evidence of tuberculosis in 26% of 230 cases certified as " silicosis " during life. In 1949, McVittie reported a similar finding (30% positive). Rogers (1946), on the other hand, reported the presence of tuberculosis in 75% of nearly 1,000 cases. It is difficult to account for the different findings of these workers but it is possible that it is caused by differences in the selection of the cases examined.

Similar divergent results are found in classical silicosis. Meiklejohn (1949 a and b) reviewed the literature and quoted that the proportion of cases complicated by tuberculosis varied from 46% to 75%; in his own series of 750 necropsies on pottery workers he found 40% of the cases of silicosis to be positive (only 36% of the cases of advanced silicosis were positive).

Recently James (1954) reported a series of 454 cases of massive fibrosis with evidence of tuberculosis (usually histological but sometimes including demonstration of the bacillus) in 40%, and also drew attention to the higher proportion of young men whose lesions were complicated by tuberculosis as compared with the older men. Of 17 cases under the age of 40, 15 were positive (88%), whereas in those aged 60 and over active tuberculosis was found in only 32 out of 112 cases (29%). Our series is too small for analysis by age groups. This relationship of the prevalence of tuberculosis to age at necropsy may well be due in part also to the nature of selection by death, whereby the more fatal type of disease is predominant in the younger age groups. On account of the many factors which may influence the selection of post-mortem material one cannot draw any firm conclusions from the age distribution of tuberculosis at necropsy as to the association between such tuberculosis and the pneumoconioses during life. Nevertheless, Carpenter, Cochrane, Jarman, and Miall (1956) note the decline of the prevalence of positive sputum with increasing age in cases of massive fibrosis in the Rhondda Fach.

Tubercle bacilli are found in the sputum of only a small proportion of cases of massive fibrosis during life. In the Rhondda Fach Survey Cochrane *et al.* (1952) found only 1·1% positive by laryngeal swabbing; in contrast 2·29% of apparently healthy women between the ages of 20 and 25 were positive. In cases of massive fibrosis studied as in-patients of the Pneumoconiosis Research Unit 7·7% showed tubercle bacilli in sputum examined by culture and

guinea-pig inoculation (Kilpatrick, Heppleston, and Fletcher 1954). Our results show that cultures taken from the bronchus before the lung had been incised at necropsy yielded 33 (28%) positive (see Table 2), and it would seem probable that if a

TABLE 2

ISOLATION OF TUBERCLE BACILLI FROM 41 CASES OF PROGRESSIVE MASSIVE FIBROSIS

Group	Total Positive	No. Positive by Method of Examination		
		Culture of Swab of Bronchus	Culture of Lung or Gland	Guinea-pig Inoculation of Lung or Gland
P.M.F.	8	2	7	8
P.M.F. with obvious tuberculosis	31	29	30	31
P.M.F. with suspicion of tuberculosis	2	2	2	2
Totals ..	41	33	39	41

specimen of sputum had been examined shortly before death it also would have yielded tubercle bacilli. This is supported by the results in the hospital population which indicate an association of "illness" with increased prevalence of tuberculosis. The prevalence of tuberculosis in patients admitted to hospital lay between that found in patients living at home and that found at necropsy.

Table 3 has been added to show that there are a large number of cases of massive fibrosis with

TABLE 3

CAVITATION AND/OR NECROSIS IN CASES OF PROGRESSIVE MASSIVE FIBROSIS

Isolation of Tubercle Bacilli	Cavitation and/or Necrosis	No Cavitation and/or Necrosis
Positive	36 (40%)	5 (17%)
Negative	53 (60%)	24 (83%)

cavities which do not yield tubercle bacilli. A similar observation has been made in a hospital population during life (Kilpatrick *et al.*, 1954).

There is undoubtedly a high prevalence of tuberculosis in the cases of massive fibrosis which come to post-mortem examination, and it is not clear if this is a breakdown of an old tuberculous infection which was necessary to the formation of the lesion, or a recent infection by tubercle bacilli into a previously non-tuberculous massive fibrous lesion.

Finally, our results show that more careful and detailed search for tubercle bacilli in the massive lesions did not yield a higher proportion of positive cases than had been previously reported.

Summary

One hundred and fifty three cases of coalworkers' pneumoconiosis were examined at necropsy for the presence of tubercle bacilli both by culture and by animal inoculation. The series contained 118 cases of progressive massive fibrosis which were the special object of this study. Tubercle bacilli were isolated from 41 cases (35%) of massive fibrosis, and in 31 (28%) of these naked-eye or histological evidence of tuberculosis was recognized pathologically. The pathological observations, therefore, correlated well with the bacteriological results. There is a marked difference between the prevalence of tuberculous infection found in a post-mortem series and that found during life in cases of progressive massive fibrosis. During life the observed prevalence is only 1·1%. We found tubercle bacilli in the bronchi at necropsy in 28%. Thus open tuberculosis is, in most cases, a late complication of massive fibrosis. Whether or not tuberculous infection occurs early or late cannot be deduced from this study.

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THE INFLUENCE OF THE SHAPE OF DUST PARTICLES ON THE RATE OF PHAGOCYTOSIS *IN VITRO*

BY

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Phagocytosis of the dust particles is one of the most striking phenomena observed in histological sections of lungs exposed to the effects of dusts. Therefore, a number of authors who tried to explain the mechanism of the origin of silicosis paid special attention to the question of phagocytosis.

Some of them were of the opinion (Fenn, 1922) that the rate of phagocytosis was indirectly proportional to the danger of various kinds of dusts. Fenn's results support this hypothesis as far as the effects of coal dust and quartz are concerned. On the other hand, Franks and Watt (1934) and Kasten (1939) found that these two kinds of dusts of biologically different efficiency were phagocytosed, roughly in the same degree, in an *in vitro* experiment. Further experiments by Policard (1948) have not led to general agreement on this problem. The lack of agreement may be caused partly by the variety of tissues used (embryonic lung tissue, spleen, suspensions of leucocytes), partly by differences in the state of the dusts, or perhaps to inadequacy in the methods of estimating the degree of phagocytosis.

Recent work shows that in addition to the effect of the chemical properties of the dust some physical properties may to some extent control its biological efficiency (King, Mohanty, Harrison, and Nagelschmidt, 1953). Some of the later work supports the hypothesis propounded by Fenn (1922) on the indirect relation between the " harmfulness " of dusts and the degree by which they are phagocytosed. Thus Jötten and Marwyck (1952) showed that protective dusts, which in animal experiments diminish the silicotic effectiveness of quartz, increase at the same time the efficiency by which quartz is phagocytosed in experiments *in vitro*. The knowledge of factors which may determine the rate of phagocytosis of quartz dusts appears therefore to be of great importance, and the shape of the quartz particles may be one of these determining factors.

Material and Methods

Our first objective was to ascertain the degree of dependence of the rate of phagocytosis on the shape of phagocytosed particles. The shape of particles was characterized by the relation of the minimum to the maximum dimension of their projection under the microscope, and each of these extreme dimensions had its own distribution in the given sample of dusts. An ideal arrangement of the experiment would have been one in which the rate of phagocytosis of particles for different ranges of ratios of extreme dimensions could have been ascertained. Since, however, such an experiment was not feasible, we proceeded in such a way that the particles were distributed into two groups: (1) the " long " particles where the length exceeds the width, and (2) the " round ones ", where both of these dimensions are approximately equal. Since such a determination can to a certain extent be considered subjective, a control was established by repeated classifications of particles from different sub-samples taken from the same principal sample. The ratios between the number of particles of the first and the second group in each sub-sample agreed well.

The study was carried out in cultures of macrophages derived from the spleen of 14-day chicken embryos cultivated in hanging drops. The degree of phagocytosis was evaluated with a phase-contrast microscope.

Reproducible doses of dusts were achieved by methods already described by Lengerová, Lenger, Esslová Tuscany, and Volfová (1956). The cultures were made on coverslips, dust being suspended in a diluted embryonic extract. Instead of normally hollow-ground slides, glasses with planparallel bottoms, which do not disturb observations through the phase-contrast microscope, were used. The distribution of the particle sizes was such that 99·4% did not exceed 5μ.

For both shapes investigated the relation, $Z = \frac{N_r}{N_t}$, *i.e.*, the relation between the number of the long and the round particles taken from a large number of particles (15,000) was first estimated. With the experimental cultures, this relation was checked once more by counting 1,500 particles before inserting the cultures in the thermostat (t value).

The methods of quantitative evaluation of phagocytosis *in vitro* consist in ascertaining the number of particles phagocytosed per phagocyte (Fenn, 1922; Jötten and Marwyck, 1952). In our experience, however, this procedure is unreliable, because, even when phase contrast microscopy is used, the number of phagocytosed particles of SiO_2 cannot always be determined reliably; quartz particles within the cell gradually lose their sharp edges and after 24 hours the determination of the number of phagocytosed particles in the critical range up to 5μ is very unreliable. Thus an indirect procedure was used which is based on the determination of the decrease of the number of quartz particles in the zone of emigration of phagocytosing cells compared with the initial value at t°. A possible objection to this procedure is that this decrease will include also those particles not phagocytosed but merely covered with macrophages. It was found, however, that with a suitable density of particles and macrophages this error was negligible. Moreover, the probability of the long or round particle being covered corresponds to the proportion of that particle in the sample, and, therefore, the fundamental relation N_r/N_l is not changed.

With all cultures of macrophages the relation N_r/N_l was estimated before inserting the cultures into the thermostat, and then during the incubation at four-hour intervals up to 24 hours. The evaluation of the number of particles was always carried out in the same sphere round the mother fragment, into which the macrophages emigrate, and that in the microscopic plane with the maximum number of macrophages. Some observations were made in which the cultures were followed throughout 24 hours of the experiment, a heated stage and phase-contrast equipment being used. Other cultures were fixed at different intervals with Carnoy's fluid, then mounted on Canada balsam, the decrease of particles being evaluated again by means of the phase-contrast microscope. For the different time periods the relation of N_r/N_l was always based on examining an area in which there were about 500 macrophages.

Results

The time course of the relation of N_r/N_l for quartz (curve 1) and for silicon carbide (curve 2) is shown in Fig. 1. During the first four hours of the experiment, when the emigration of macrophages is slight, the initial ratio remains practically unaltered and with SiO_2 approximately equals 2 in favour of round particles. Between the fourth and the sixteenth hours the value N_r/N_l gradually decreases. About the tenth hour the value begins to decrease until it is below 1. After 16 hours it again rises and at the end of the experiment, *i.e.*, after 24 hours, the value 1 of N_r/N_l is again reached.

For the dust of silicon carbide (curve 2, Fig. 1) the course of the relation N_r/N_l is analogous but not so striking, *i.e.*, with SiC the fundamental relation N_r/N_l was just a little higher than 1 at the start and its decrease with time was less marked;

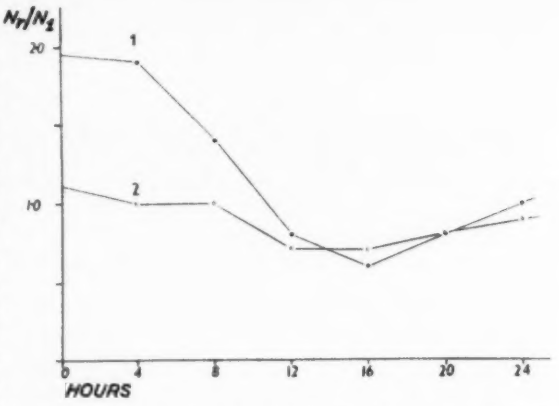

FIG. 1.—The time course of the relative values N_r/N_l throughout the duration of the experiment.

the same value as for SiO_2, however, was nearly achieved, after which it began to return to 1 like quartz.

The real phagocytic activity of macrophages is not evident from Fig. 1, and that is why in Fig. 2 the decrease of an absolute number of SiO_2 particles is shown in the mean field of view during the 24-hours experiment, which is the result of the phagocytic activity of macrophages. Curve 1 refers to all SiO_2 particles irrespective of shape distribution, curve 2 to the round particles, and curve 3 to the long ones. Fig. 3 demonstrates the velocity of phagocytosis of quartz particles as a function of time. Based on the experimental values, the velocity,

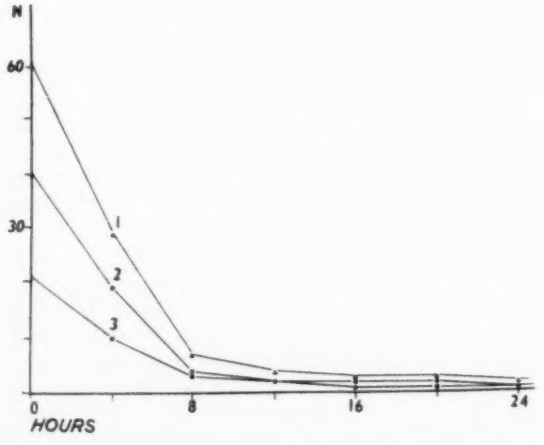

FIG. 2.—The decrease of an absolute number of quartz particles in the mean field of view during 24 hours of the experiment. Curve 1 represents SiO_2 particles irrespective of shape distribution, curve 2 round particles, and curve 3 long particles.

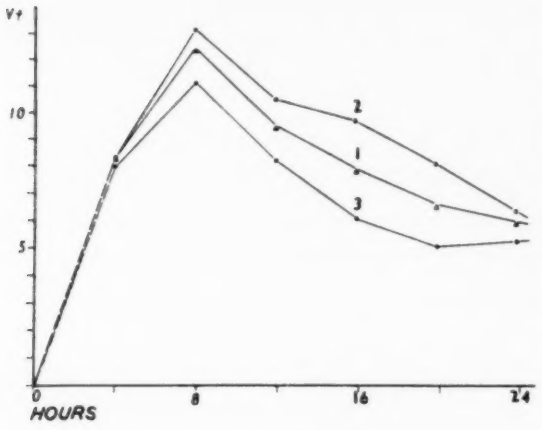

FIG. 3.—The velocity (V_f) of phagocytosis of quartz particles as a function of time (t in hours).

V_f, is calculated from the relation given by Fenn (1921)

$$V_f = (1/t)\log \frac{A}{A - X}$$

where A = the number of particles in the field of view, X = the number of phagocytosed particles, t = time.

It is quite evident that the velocity of phagocytosis of the round particles (curve 2) is higher than that of the long ones (curve 3), both reaching the maximum about the eighth hour of the experiment when the decrease occurs, and the values are almost the same for the remaining time, *i.e.*, up to 24 hours. With SiC the ratios are similar. The decrease of the velocity of phagocytosis (Fig. 3) agrees in time with the decrease of an absolute number of particles in the field of view (Fig. 2), which points to the fact that the cause of the decrease of the phagocytic velocity is due to the decrease of the probability of a collision between a macrophage and a particle. In an attempt to interpret the curves obtained in Fig. 1 we have started from this premise. If the shape factor had no influence on the rate of phagocytosis of dust particles, the probability of phagocytosis of different extreme types of shape of particle would be proportional only to their presence in the sample of dust. Thus an absolute number of particles would decrease with the course of the experiment but the relative presence of both types of particle would remain constant. The course of the value N_r/N_l would then be expressed by a straight line parallel to the axis x. In fact, the value N_r/N_l decreases till under the value 1, which means that with time the round particles decrease proportionately more rapidly than the long particles. While at the beginning of the experiment there were twice as many round quartz particles as long ones, after 16 hours there was a larger number of long particles than round ones in the area of emigrated macrophages. Thus the round particles were phagocytosed preferentially. If we accept this opinion the fact that the increase of the value N_r/N_l (Fig. 1) recurs after 16 hours of the experiment seems to be striking; this increase does not exceed the value 1. Nevertheless, it seems to point to the fact that between the sixteenth and twenty-fourth hours it is again the long particles which are phagocytosed more effectively. We believe that this phenomenon may be explained by the majority of dust particles in the zone having been phagocytosed (Fig. 4) so that the error of estimating the ratio is large (Fig. 2).

We consider, therefore, that the first part of the curve in Fig. 2, proving the selective phagocytosis of round particles, is of great importance. This phenomenon cannot be explained to our satisfaction by any of the existing theories of phagocytosis. The influence of the shape could be important if phagocytosis of SiO_2 particles consisted of a chemctactic action of impurities adsorbed on the surface, which corresponds to McCutcheon's hypothesis (1942). The surface of the long particle is, roughly, 50%

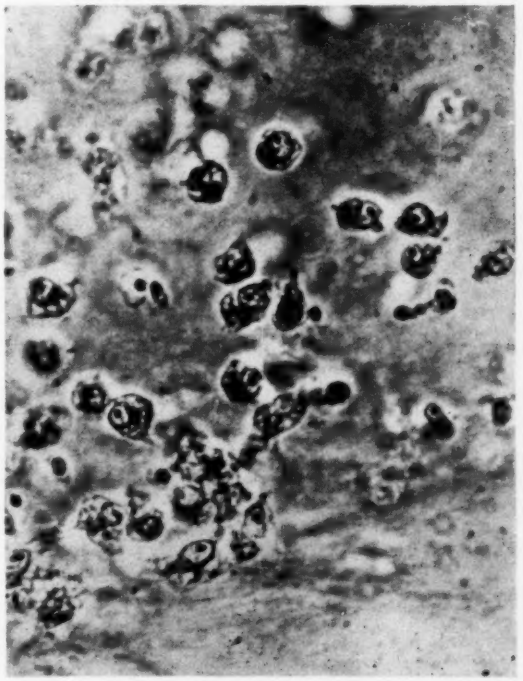

FIG. 4.—Photomicrograph of silicon carbide particles after 24 hours of the experiment. Most of the dust particles are already phagocytosed by the phagocytosing elements.

greater than that of the round particle for the same volume, and consequently the long particle carries a greater quantity of absorbed impurities. As our experiments show that the long particles are substantially phagocytosed more slowly than the round ones, we should have to suppose (McCutcheon's hypothesis, 1942) that the activity of the adsorbed impurities is chemotactically negative to the phagocytosing elements.

It is also interesting to consider the influence of shape in relation to the findings of King and Nagelschmidt (1954) that the solubility of silicic acid represents the function of the specific surface and that the greater the surface the greater the fibroplastic effect. In this case, the long particles would again represent the particles of greater specific surface, which would therefore release more silicic acid and show a greater biological activity. In summary, it would mean that the particles of a certain shape might be biologically more active and at the same time be less readily phagocytosed, and so, according to the views of Fenn (1922), Lauche (1931), Jötten and Marwyck (1952), and others, more harmful. Further experiments are necessary to prove this hypothesis when estimating the harmfulness of different dusts chemically identical.

Summary

The rate of phagocytosis of two shapes of dust particles—" long " and " round "—was studied in an investigation *in vitro* in cultures of macrophages from the spleen of chicken embryos. The round particles were phagocytosed preferentially. This result is discussed from the standpoint of existing theories of phagocytosis as well as from that of the relation between the rate of phagocytosis and the harmfulness of different kinds of dusts.

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THE TOXICITY OF FUMES FROM A DIESEL ENGINE UNDER FOUR DIFFERENT RUNNING CONDITIONS

BY

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It has been suggested that fumes from diesel engines make an important contribution to atmospheric pollution. This is probably because they are sometimes highly irritant and sometimes very smoky. It is well known that, because of their comparatively low carbon monoxide content, these fumes are much less lethal than those from petrol engines.

The leading work on diesel fumes has been done by the U.S. Bureau of Mines. Holtz, Berger, Elliot[1], and Schrenk (1940) analysed the fumes from engines in proper mechanical conditions, plotting the carbon monoxide, nitrogen oxides, and aldehyde content against fuel-air ratio.

The present work was directed to ascertaining what were the main toxic constituents of diesel fumes. In order to do this animals were exposed to the fumes and at the same time the concentration of various constituents of the fumes was determined; pathological investigations were subsequently carried out on the exposed animals.

In this paper nitric oxide is designated NO, and nitrogen dioxide NO_2. For conversion of parts per million to mg./m³ factors valid at 20°C. have been used.

Apparatus and Materials

Engine.—The engine used was a Royal Enfield single cylinder air-cooled diesel engine. Detailed features of this engine are:—

Cylinder bore	3·346 in. (85 mm.).
Cylinder stroke	3·937 in. (100 mm.).
Cylinder capacity	568 ml.
Compression ratio	19·5 to 1.
Valve gear	Overhead operated by push rods.
Fuel injection	C.A.V. equipment operated from engine camshaft.
Atomizer	C.A.V. single hole type with "leak off" connexion.
Governor	Centrifugal type mounted on end of camshaft. Adjustable for any speed between 1,200 and 1,800 r.p.m.
Power developed	3·75 to 6·0 B.H.P. at 1,200 to 1,800 r.p.m.
Fuel consumption	0·42 pints/hr. (570 ml./hr.) at 1,500 r.p.m.

Fuel.—The fuel used was diesel fuel, 47 cetane, obtained from the Regent Oil Co. Ltd. It had been produced by distillation from Venezuelan mixed base crude. Its characteristics were as follows:—

Viscosity No. 1 Redwood at 100°F. (37·8°C.) (kinematic viscosity approx. 3·0 centistokes)	33 sec.
Specific gravity at 60°F. (15·6°C.)	0·838.
Flash point	210°F. (99°C.)
Neutralization number (total acidity)	0·1136 mg. KOH per gm. oil
Carbon (Conradson)	0·006%
Pour point	Below —30°F. (—34·4°C.)
Water content	Nil

Distillation range (10 mm. Hg)

	Initially	10%	20%	30%	40%	50%	60%	70%	80%	90%
°F	130°	184°	215°	235°	262°	283°	295°	321°	347°	371°
°C	54·5°	84·5°	102°	114°	128°	140°	146°	166°	175°	189°

Sulphur content .. 0·51% (determined by Institute of Petroleum lamp method)

Exposure Chamber.—The exit of the silencer of the engine was connected by a flexible metal pipe, 695 cm. long and 3·2 cm. in internal diameter, to a 10 cubic metre chamber. Two exhaust pipes from the chamber led to the outside air. The contents of the chamber were circulated by means of a fan. The temperature of the chamber during a run was 2-3°C. higher than that of the outside air. The walls of the chamber were of glass and painted steel; the floor was of concrete.

It is to be noted that in toxicity experiments with nitric oxide present the size of the chamber and the flow rate through it may have a considerable effect on the results. The reason is that the nitric oxide is slowly oxidized to the dioxide which is relatively more toxic. Hence if fumes of constant nitric oxide content are supplied to a chamber, the toxic effects on animals exposed in it will be greater the lower the rate of change of atmosphere in the chamber. In this case the fumes were supplied at a rate sufficient to fill the chamber in about 20 minutes (or 40 minutes under conditions D).

Experimental Methods

Running Conditions of Engine.—Four conditions of engine running were investigated. The essential features are given in Table 1.

Details of the running conditions are as follows:—

A. The engine was run with only its accessories,

including the cooling fan, taking power. Under these conditions the exhaust was acrid (causing lachrymation in under 10 seconds). At times it was almost clear, but sometimes white fumes were produced; the visibility in the chamber was then about 6 ft.

B. To the engine were coupled a large fan and two hydraulic pumps to provide a load. The fumes produced under these conditions were less acrid but more toxic than under a light load.

C. The load was arranged as under condition B but the original injector (which was in good mechanical condition) was replaced by one which was stated by the manufacturer to be " worn ". This replacement affected the fuel-air ratio but little; the acridness and toxicity of the fumes were, however, much diminished.

D. To produce a higher fuel-air ratio, the air inlet connexion was partly closed with a metal blank. The load consisted only of the accessories. The area covered was adjusted by experiment to give continuous white smoke from the exhaust. The open circular segment had an area of approximately 0·03 sq. in. (0·193 sq. cm.) as compared with the normal inlet area of 1·227 sq. in. (8·6 sq. cm.). Under these conditions the engine produced a dense white smoke, apparently consisting mainly of unburnt oil; very little carbon was present. The fumes were very acrid, causing intense pain to the eye in 4 to 7 seconds. The visibility in the chamber was only a few inches, and the lethal quality of the fumes was greater than under conditions A, B, and C. In the early stages of running under these conditions the firing was irregular, as if on some strokes the engine failed to fire; it grew more regular as the engine warmed up.

Sampling and Analysis.—To obtain an accurate assessment of the dosage to which the animals were exposed, all samples, other than that for total oxides of nitrogen, were collected at a constant rate throughout each experiment.

Samples of gas for CO and O_2 determination were taken by drawing fumes into a balloon at a uniform rate by means of a suction device. Carbon dioxide samples were collected by drawing the fumes through bubblers at 50 ml./min. by means of an aspirator. Other samples were collected in bubblers at 1 l./min., critical orifices being used to control the flow. To protect the orifices and balloons from dirt, the fumes were sucked through two Whatman No. 1 filter papers in series before entering the bubblers. The total particulate matter, black smoke, particulate acids, and vanadium contents of the samples collected on these papers were determined.

In every case two bubblers and two filter papers were used in series. Determinations were carried out on each separately, and the measured concentration was corrected for slip.

The fuel-air ratio was obtained by measuring the fuel consumption and (when the air intake was unobstructed) calculating the air intake from the cylinder volume and engine speed. A volumetric efficiency of 90% was assumed. Under conditions D the air intake was measured directly using a " rotameter " flowmeter.

Determinations of Oxygen, CO_2, and CO.—Oxygen was determined in a gas sample by Haldane's method, carbon dioxide by absorption in N/2 sodium hydroxide which was then titrated with acid using phenolphthalein and methyl orange as indicators, and carbon monoxide from a gas sample by means of the Grubb Parsons infra-red gas analyser.

Determination of Aldehydes.—Aldehydes were determined by absorption in hydroxylamine hydrochloride solution which was initially adjusted to *p*H 4·5. The aldehydes form oximes with the hydroxylamine, liberating hydrochloric acid, which is then titrated.

As the hydroxylamine hydrochloride has a buffering action, the concentration used must be chosen to suit the quantity of aldehyde to be determined; a large excess of the former must not be used. (Under conditions D insufficient hydroxylamine was used, and the concentration of aldehyde had to be determined in a subsidiary experiment.) This method is also sensitive to certain ketones, including acetone. Control bubblers containing distilled water were used to obtain a correction for acids directly absorbed from the fumes.

Total Particulate Matter.—This was determined by weighing the filter sample; the paper was previously weighed, a control filter paper being used as a counterweight to compensate for variations in atmospheric humidity.

Particulate Acids.—These were determined by titration of a filter sample at *p*H 4·5; they were expressed as sulphuric acid, but other acids such as formic and acetic may have been present.

Black Smoke.—Black smoke was determined by comparing the shade of the stain on the filters with those of a series of standard stains obtained from an atmosphere of smoke from a kerosene lamp. The concentration of smoke in this standard atmosphere was determined by weighing samples. As different smokes differ in blackening power, this method can give qualitative results only.

Vanadium and Nitrogen Dioxide.—It was shown by the method of Woodman and Cayvan (1901) that the concentration of vanadium in the fumes was less than 10 μg./m^3, if indeed it was present at all. The determination of nitrogen dioxide (NO_2) in the presence of nitric oxide was difficult. Holtz *et al.* (1940) determined only total oxides of nitrogen. Various methods were tried; values obtained by means of the optical absorptiometer of Sinkinson (1954) were found to be compatible with those obtained by absorption in alkali in two large sintered glass bubblers followed by determination of nitrite by the Griess-Ilosvay reaction. The mean of the values obtained by these two methods was used.

It was found that if a long train of bubblers is used oxidation of NO to NO_2 causes errors.

Total oxides of nitrogen were determined by admitting fumes to an evacuated bottle containing alkali. This was left overnight to allow complete oxidation of NO, and nitrite was determined by the Griess-Ilosvay reaction.

A colorimetric method for determining sulphur dioxide, based on its reaction with decolorized fuchsin, failed to give positive results owing probably to the presence of oxides of nitrogen. To obtain a value for the maximum possible sulphur dioxide content of the fumes under

conditions A a known volume of the fumes was bubbled through hydrogen peroxide (1 vol.), initially at *p*H 4·5, the acid produced being titrated.

Toxicity Experiments.—Preliminary experiments showed that the toxicity of the exhaust fumes varied somewhat from day to day even if no deliberate alteration to the running conditions was made. It was, therefore, necessary to repeat the whole series to see whether results reproducible enough to show any alteration in toxicity with running conditions could be obtained. In fact, while the mortalities under a given set of conditions were not always the same in the two series, the difference between them was much less marked than that between the mortalities under different running conditions. In both series the arrangement of running conditions in order of lethality was D, B, C, A. In Table I the mortalities in the two series are (where they differed) given separately.

It was found that animals could usually be exposed to undiluted diesel fumes for some hours without asphyxia.

Two series of experiments were performed. In each set eight rabbits, 20 guinea-pigs, and 80 mice were placed at random into four batches. Each batch was exposed in the chamber to undiluted diesel fumes for five hours or until all the animals were dead. Each of the four batches was exposed to fumes produced under one of the four running conditions described above. The whole experiment was then repeated using four batches from another set of animals. In Table I the results of these two sets are combined, but some individual experiments are referred to below as A1, A2, etc.

In an experiment, the engine was first started and left on for half an hour so that the chamber could fill with fumes. (The flow of fumes at room temperature was about 500 l./min. under conditions A, B, and C and about 250 l./min. under D.) The animals were then placed in the chamber in cages and sampling was started. At intervals the animals were inspected and any dead were removed. After five hours the engine was stopped and the chamber was cleared.

The next day, if there had been no mortality in any one of the species, one of the species was killed with nembutal and a post-mortem examination was performed.

Three weeks after each experiment on the first set, and one week after each experiment on the second set, all the surviving animals were killed with nembutal and examined at necropsy.

In all cases specimens of lung, trachea, and other tissues were taken from representative animals and sections were stained with haematoxylin and eosin. In cases where carbon monoxide poisoning was suspected (conditions D) carboxyhaemoglobin was determined at necropsy by colour comparison using a Lovibond's comparator.

Lachrymation time was measured by an observer applying his eye to a port in the chamber wall and noting the time required to produce intolerable irritation.

Results

The mortality data and the concentrations of the more significant components are summarized in Table 1. This gives the mean of all the reliable determinations made under given running conditions. The total mortalities are also summarized. The less important constituents are discussed below.

The oxygen concentration was above 16% in all cases except in D2, when it was 11·4%. Carbon dioxide ranged from 2·34% to 3·58%. Particulate acids were equivalent to less than 1 p.p.m. of sulphuric acid in all cases. The maximum black smoke content found was 2·2 mg./m³ (conditions C), but this determination is, as explained, qualitative only. Gaseous acids were found equivalent to 17 p.p.m. of sulphur dioxide at the exit of the silencer, and 5·8 p.p.m. in the chamber. The vanadium content of the fumes was less than 10μg./m³.

The following points not given in Table 1 are worthy of record:—

In no case did the fumes appear black or smoky. The liquid collected from them appeared to be unburnt diesel oil. A smoky exhaust, such as is frequently criticized as a source of atmospheric pollution, was not used in this series of experiments.

The subjective differences in the irritancy and lachrymating power were greater than might appear from the measured lachrymation times. Under condition D the fumes were violently irritant, and produced severe and lasting pain in the eyes. The change in irritancy on replacing the "good" injector (B) by a "worn" one (C) was very marked. When the latter was used the fumes were almost non-irritant. Likewise the fumes under condition A were definitely more irritant (though less toxic) than those under condition B.

There are significant differences between the mortalities recorded with the same conditions and animals from different batches. In conditions B and C, deaths usually took place in the last hour of the five-hour exposure, or on the following day. In conditions D, the mice died first; they died more rapidly in D1 than in D2. After one hour's exposure in D1 (0·22% CO), all except three of the 20 mice were dead. All the rabbits and guinea-pigs were alive. After 3 hr. 20 min. all the animals were dead and the experiment was stopped. In experiment D2 (0·12% CO), on the other hand, one mouse only was dead at the end of an hour. After 3 hr. 20 min. 19 mice and one guinea-pig were dead, but both rabbits were still alive. After 4 hr. 35 min. all the animals were dead.

Post-mortem Findings: Macroscopic.—One of each from the animals exposed under conditions A was killed with nembutal the day after exposure. In general the appearance of the lungs was normal but in one mouse there was congestion of a hepatic type. As there were no fatalities in this group it seems

TABLE 1

SUMMARY OF EXPERIMENTS ON EXPOSURE OF ANIMALS TO DIESEL FUMES

Running Conditions	A	B	C	D
Speed (r.p.m.)	1,600	1,600	1,600	1,600
Power output (kw.)	Low	4·1	4·1	Low
(B.H.P.)	Low	5·5	5·5	Low
Fuel-air ratio	0·0121	0·0193	0·0199	0·0333
Injector condition	Good	Good	Worn	Good
	Open	Open	Open	Restricted
Composition and Properties				
CO (%)	0·056	0·041	0·038	0·17
NO_2 (p.p.m.)	23	51	43	12
Oxides of N (as NO_2) (p.p.m.)	46	209	174	44
Aldehydes (as formaldehyde) (p.p.m.)	16	6·0	6·4	154(*c*)
Particulates (mg./m³)	74	122	53	1,070
Visibility (ft.)	6	1 to 6	6	0·5
Lachrymation time (sec.)	7·5	10	20	5·5
Animals				
Duration of exposure (hr.)	5	5	5	5
Mortality (*a*) during exposure (*b*) total (up to 7 days)				
Out of 40 mice	(*a*) 0 (*b*) 0	(*a*) 13 (*b*) 19 (*d*) 10;3 (*d*) 13;6	(*a*) 0 (*b*) 1 (*d*) 0;0 (*d*) 1;0	(*a*) 40 (*b*) 40
„ „ 10 guinea-pigs	(*a*) 0 (*b*) 0	(*a*) 0 (*b*) 9 (*d*) 0;0 (*d*) 4;5	(*a*) 1 (*b*) 6 (*d*) 0;1 (*d*) 1;5	(*a*) 10 (*b*) 10
„ „ 4 rabbits	(*a*) 0 (*b*) 0	(*a*) 0 (*b*) 0	(*a*) 0 (*b*) 0	(*a*) 4 (*b*) 4
Pathology				
Tracheal damage	Mild	Nil	Nil	Nil (mice) Severe (others)
Lung damage	Mild	Severe	Moderate	Nil (mice) Mild (others)
Carboxyhaemoglobin	Low	Low	Low	60% (mice) 50% (others)
Most sensitive animal	Guinea-pig	Guinea-pig	Guinea-pig	Mouse
Postulated main cause of death:				
Mice	(1) Irritants (*e*) (2) NO_2 (*e*)	NO_2	NO_2	CO
Guinea-pigs	(1) Irritants (*e*) (2) NO_2 (*e*)	NO_2	NO_2	(1) CO (2) Irritants
Rabbits	(1) Irritants (*e*) (2) NO_2 (*e*)	—	—	(1) CO (2) Irritants

(*c*) Determined in a subsidiary run
(*d*) Mortalities in the two series are given below the total mortality in each case
(*e*) In exposures of seven or 14 hours

likely that this mouse would have recovered. Animals killed one or three weeks after exposure under conditions A sometimes showed emphysematous raised areas or patches of congestion in the lungs but were usually normal in their post-mortem appearance.

In the animals which died after exposure under conditions B, there was severe pulmonary damage. The greatest damage was found in those animals which survived the exposure and died the next day. Nearly always there was severe oedema with frothy fluid oozing from the cut surfaces of the lung. Often there were alternating patchy areas of consolidation and oedema. In many cases froth oozed from the trachea. The blood was normal in colour, even in animals dying in the fumes. The carboxyhaemoglobin content was low. The stomachs of the guinea-pigs were normal.

Similar findings were made in animals dying under conditions C; in some guinea-pigs alternating patches of consolidation, congestion, and emphysema gave the lungs a " tortoiseshell " appearance.

The lungs of animals surviving exposures under B and C conditions, and killed one or three weeks later, sometimes showed evidence of previous injury. In two mice killed three weeks later there were sharply delimited patches of consolidation at the base of the lung. Another mouse had widespread congestion of the lungs; another had complete consolidation of the right lung. In many cases, however, the lungs of survivors were of normal appearance.

The blood of the animals which had died under conditions D was scarlet. The carboxyhaemoglobin content in the mice was (by Lovibond's method) about 60%, and in the other animals about 50%.

The lungs of the mice which had died under conditions D were of normal appearance.

The guinea-pigs and rabbits which died under these conditions showed varying amounts of emphysema and congestion. One rabbit showed oedema. All the guinea-pigs showed petechial and confluent haemorrhages of the stomach with desquamation of the mucosa, and one of the rabbits showed scattered discrete small haemorrhages in the cardiac region of the stomach. One of the rabbits showed many small haemorrhages of the thymus.

Post-mortem Findings: Microscopic.—Those animals which died during the exposures all showed some degree of pulmonary abnormality. Moderate to intense capillary and venous congestion were constant findings. In the mice which had died under conditions D, the lung damage consisted mainly of intense congestion and small capillary haemorrhages, probably pre-terminal.

The degree of lung damage was rather greater in the guinea-pigs and rabbits from the D exposures than in the mice.

Animals which died during the B and C exposures all showed more severe lung damage. Oedema was a fairly constant feature, more marked in the guinea-pigs than in the mice; of the latter a few showed traces only of oedema. In those cases where consolidation was present this was invariably associated with alveolar collapse and oedema. Emphysema was also present. In the mice, damage to the bronchial and bronchiolar epithelium was slight, whereas the guinea-pigs showed commencing bronchiolitis.

Animals which died after removal from the fumes invariably showed much more severe lung damage than those which died during the experiment. This is to be expected, as not only was exposure longer for the survivors but the delayed effects of the nitrous fumes had more time to develop. In the guinea-pigs in particular there was severe oedema.

Animals killed the day after the experiment (because no member of their species had died) showed little damage. Mice exposed under conditions A had slight oedema but in general the lungs were almost normal. Guinea-pigs had some patchy consolidation of the lungs with some emphysema. There was rather more damage in experiment A2 (22 p.p.m. aldehyde) than A1 (9·9 p.p.m. aldehyde).

The animals killed seven or 21 days after the experiment showed varying stages of repair.

The tracheas of the animals in experiments B and C showed little damage, and the same was true of the larger bronchi. Slight damage was evident in experiment A; a rabbit exposed in A1 and sacrificed the next day also showed desquamation of the epithelium of the trachea.

The larger bronchi of the mice in experiment D showed no damage, but the tracheas and bronchi of the guinea-pigs and rabbits in experiment D showed very serious damage indeed. In one guinea-pig the whole of the tracheal epithelium had been removed and there was evidence of formation of a false membrane. In one rabbit the thymus was found to be haemorrhagic. The abnormality appeared to be vascular engorgement with many small capillary haemorrhages.

Subsidiary Experiments

Acrolein.—Acrolein was used to determine the pathological effects of a typical organic irritant.

Two rabbits (2·6 and 2·8 kg.), four guinea-pigs (mean weight 228 g.), and 20 mice (mean weight 20 g.) were exposed for six hours to acrolein at a mean concentration of 10·5 p.p.m. (24·4 mg./m^3). The total dosage was 8,790 mg.min./m^3. At the close of the experiment all the animals were alive; the mice were in a very somnolent condition, as if they had had an insufficient dose of anaesthetic. One guinea-pig and two mice were killed for post-mortem examination: another guinea-pig died shortly afterwards and another overnight. Eight mice died the next day, and a further mouse the day after. The mortality was 1/2 for rabbits, 2/4 for guinea-pigs, and 9/20 for mice.

In the one guinea-pig and two mice killed immediately after the exposure there was macroscopically little evidence of abnormality. A clear frothy fluid was present in the guinea-pig's trachea. Sections of the lungs showed slight changes only; there was acute emphysema and in the guinea-pig patchy areas of oedema. The epithelium of the bronchi and bronchioles was intact and showed slight increase in its mucous covering.

The guinea-pig which died shortly after exposure had lungs which were voluminous but of almost normal appearance. Sections showed widespread emphysema with some partial consolidation, oedema, and congestion. Slight inflammatory cell infiltration was present. Another guinea-pig dying overnight had both upper lobes of the lungs completely consolidated. Microscopically there were acute emphysema, widespread oedema, and generalized capillary congestion. The trachea showed severe damage with much desquamation of the epithelium. In the lumen was found almost non-cellular, partly fibrinous, exudate. There was a moderate degree of inflammatory cell reaction; the picture was one of acute desquamating tracheitis.

Mice dying the day after exposure showed gross pulmonary changes. In the trachea of one of them the epithelium had been replaced by a cellular exudate, mainly of inflammatory blood cells. In the lungs there were consolidation, congestion, and inflammatory cell reaction. In the rabbit which died 36 hours after exposure, there was found generalized emphysema with patchy areas of congestion and consolidation. The epithelium of the bronchi had desquamated and some bronchi were partly plugged.

The picture of acrolein poisoning is, therefore, one of gross damage to the trachea, bronchi, and lungs. This contrasts with the action of diesel

fumes under conditions B and C, where damage to the trachea was slight. Deaths which were delayed for more than 24 hours after exposure occurred with acrolein, but not with diesel fumes.

Diesel Fumes (Long Exposures under Conditions A).—As five hours' exposure produced no fatalities, the results of two long exposures are reported.

Two rabbits, 10 guinea-pigs, and 50 mice were exposed to undiluted fumes from the engine running under conditions A (light load). On the first day the exposure time was seven hours; nine of the guinea-pigs died.

Overnight the remaining guinea-pig, one rabbit, and 10 mice died.

The next day exposure continued for a further seven hours; the remaining rabbit and 35 of the mice died during exposure.

The mortalities for the three species were therefore:—

7-hour exposure	1/2, 10/10, 10/50
14 hour exposure	2/2, 10/10, 45/50

Within half an hour of the beginning of the experiment the mice became lethargic. The behaviour of the other animals was normal.

No chemical analysis was performed during this experiment.

Macroscopically the lungs of animals which had died during or after exposure for seven or 14 hours to fumes under conditions A showed varying degrees of pulmonary congestion, consolidation, oedema, and emphysema. In guinea-pigs which had died, a frequent finding consisted of small, widespread, well-defined areas of emphysema alternately with depressed areas of consolidation. Other organs showed no constant definite abnormality, though the livers were uniformly pale and in two rabbits were beginning to show a nutmeg appearance.

Microscopically in all the animals examined there was a moderate to severe degree of tracheitis; the epithelium in parts was missing with very few basal cells remaining. Many of the remaining epithelial cells were faintly stained. Epithelium where missing was invariably replaced by a layer of densely cellular exudate, the cells being mainly polymorphs. Inflammatory cells infiltrated the submucosa and (infrequently) the cartilaginous ring. Microscopic examination of the lung sections confirmed the macroscopic findings. The constant striking features were patchy areas of emphysema and partial alveolar collapse associated with oedema and capillary congestion. Haemorrhages were not a constant finding. In all sections there was seen an acute inflammation of the entire respiratory tract. Many of the bronchi and bronchioles were plugged with cellular exudate, like that in the trachea.

One rabbit and two guinea-pigs showed early bronchopneumonia.

In the mice, the pulmonary changes were not as constant as those seen in the guinea-pigs. The epithelium of the bronchi and bronchioles was intact and inflammation, where present, was of a lesser degree than in the guinea-pigs and rabbits.

The picture was, therefore, that of gross damage to all parts of the respiratory tract.

Discussion

Composition of Fumes.—The most obvious finding concerning the composition of the fumes was that in different trials under the same running conditions the composition was different. That these variations are genuine is confirmed by the fact that in experiment D2 (with 0·12% CO) the mice took much longer to die than in experiment D1 (0·22% CO). The differences between experiments under the same running conditions are, however, less than those found under different running conditions.

Holtz *et al.* (1940), in their work on the composition of diesel fumes, adopt the " fuel-air ratio " as a standard of comparison, and we have followed their example. The " chemically correct " fuel-air ratio, at which there is just enough oxygen for combustion of the fuel, is about 0·068. In our experiments, the fuel-air ratios from 0·0119 to 0·036 were explored. The highly abnormal conditions D (air intake obstructed) were not used by Holtz *et al.*

Oxides of Nitrogen.—Holtz *et al.* (1940) found that for a given engine the total oxides of nitrogen (except N_2O) had a maximum concentration, equivalent to 340 to 590 p.p.m. (650 to 1,250 mg./m³) NO_2, at intermediate values of fuel-air ratio (0·025 to 0·045); the concentration with low fuel-air ratio (0·010 to 0·015) was less (50 to 250 p.p.m., or 96 to 480 mg./m³). This is in accordance with our finding more oxides of nitrogen under moderate load (B) than under light load (A). With the worn injector, we found less oxides of nitrogen. These workers did not determine separately the concentration of nitrogen dioxide.

Hanson and Egerton (1937) found that the ratio of NO_2 to total nitrogen oxides in the exhausts of diesel engine was higher with no load than on load; our findings are in accord.

Aldehydes.—Holtz *et al.* (1940) found that the concentration of aldehydes in the fumes decreased as the fuel-air ratio increased. This is in accord with our findings when experiments A are compared with B and C. D covers conditions not used by Holtz *et al.* It must be emphasized that the method used by us for determining " aldehydes " also determines at least some ketonic bodies, and this may be true

of other methods also. They found (Holtz *et al.* 1943) that aldehydes were higher with a " leaking " injection valve; but inspection of their tables shows that installing a valve with " lopsided " or " sticky " sprays (the latter giving a spray the angle of whose cone was less than normal) caused a reduction in the aldehyde content. The effect on aldehyde content of installing our worn injector was irregular, although the reductions in irritancy and toxicity were very marked.

Carbon Monoxide.—Our findings under conditions A, B, and C are in accord with those of Holtz *et al.* (1940) who found a minimum CO content at a fuel-air ratio of about 0·03.

Black Smoke.—Holtz *et al.* did no direct measurements of the amount of soot in the exhaust, but found smoky exhausts at fuel-air ratios above the chemically correct ratio. Very little black smoke was produced in our experiments, even under conditions D. Nearly all the particulate matter found in our experiments was of an oily nature and was not of dirty appearance.

Vanadium.—Browne (1955) states that the exhausts of gas turbines using residual oils contain 20-60 p.p.m. by weight of vanadium compounds, equivalent to perhaps 6-18 mg./m³ of vanadium. The concentration in the exhaust of our engine was less than 1/500 of this. Sjöberg (1950) found that rabbits exposed to 20-40 mg./m³ of vanadium pentoxide dust for one hour a day for eight months developed emphysema; the concentration of vanadium in our experiments was therefore a long way below the toxic level.

Browne (1955) states that the vanadium content of residual oil from Venezuela (from whence the fuel for our experiments was derived) is higher than that of other residual oils. The low vanadium content of the fumes in our experiments was probably therefore due to our fuel being distillate and not residual oil.

Sulphur Oxides.—There is a discrepancy between the amount of sulphur found in the fumes and that supplied in the fuel. With fuel of composition 84% C, 0·51% S, the fumes should contain 23 p.p.m. of sulphur dioxide or sulphuric acid for each 1% of carbon dioxide plus carbon monoxide. Our fumes contained very little sulphuric acid mist; with a carbon oxides content of 2·4% (conditions A) there should have been 55 p.p.m. of SO_2, while the measured content was 17 p.p.m. It is possible that some of the sulphur may appear in the fumes as an organic compund of sulphur, as elementary sulphur, or as carbon oxysulphide. Berger, Elliot, Holtz, and Schrenk (1943) found that the amount of sulphur in the fumes from their engine was equal to that in the fuel consumed; the discrepancy in our results is unexplained.

Irritancy.—The fumes produced in experiments D were exceptionally irritant, those in experiments A highly so. The fumes of B were irritant, while the irritation from C was only slight. These differences are borne out by the measured lachrymation times. The irritancies are roughly in line with the aldehyde contents except when B is compared with C. It has been found, however (Sim and Pattle, 1955), that aldehydes differ greatly among themselves in irritancy, acrolein and crotonic aldehyde being at least a hundred times as irritant as the corresponding saturated compounds, propionic and butyric aldehydes. Again, the fumes in experiment C2 (aldehyde content 8·3 p.p.m.) were much less irritant than those in experiment B2 (4·1 p.p.m. aldehyde). Chemical composition, as expressed in terms of broad groups of substances is, therefore, unreliable as a guide to the irritant properties of the fumes. These properties can be affected by quite subtle differences in combustion conditions, and the fact that our least irritant fumes were obtained with a faulty injector demonstrates the fallacy of supposing, as does Turner (1955), that the requirements for mechanical perfection in an engine are identical with those for a physiologically or medically harmless exhaust.

The exact nature of the irritant or irritants present cannot at the moment be stated. The known irritancy of acrolein, which caused severe discomfort at 1·2 p.p.m. (Sim and Pattle, 1955), suggests that the presence of a small amount of acrolein among the aldehydes would cause the observed effects. Bodies reacting as aldehydes, but less irritant than acrolein must, however, also be present. The irritancy of fumes containing 22 p.p.m. of aldehydes, etc., was considerably greater than that found by Sim and Pattle for 20 p.p.m. of formaldehyde. If the irritation is due to aldehydes, therefore, bodies more irritant than formaldehyde must be present.

Toxicity.—In considering which of the substances found in the diesel exhaust fumes had toxic effects the simplest case is that of the mice in experiment D. Here the high carboxyhaemoglobin value in the blood, and the virtual absence of lung damage (other than congestion, which may be caused by heart failure) point to carbon monoxide poisoning as the cause of death.

The other animals in experiment D were not free from damage to the trachea, and this may have been due to their longer survival in the fumes. In

these cases the concentration of carbon monoxide was such that the animals would probably have succumbed even in the absence of the irritant bodies; the latter may have accelerated death. Acrolein administered alone produced tracheal damage, and this or some similar body could have caused it in these cases. Nitrogen dioxide was probably not important.

The fumes in B and C were much less irritant than in A, and contained less aldehyde; but they were far more toxic than those in A. Tracheal damage was negligible. It is, therefore, likely that irritant aldehydes played little part in causing death. The high concentration of nitrous fumes and the gross oedema of the lungs found (especially when death was delayed) point to nitrogen dioxide as the cause of death in these cases. The lower toxicity in C (worn injector) must be attributed to a lower concentration of NO_2 rather than to absence or irritant aldehydes.

It may be supposed that in B and C the carbon monoxide accelerated death, and that in its absence survival would have been longer and lung damage greater than was actually observed. Bearing this in mind our results are in accord with those of La Towsky, MacQuiddy, and Tollman (1941) who found 55 p.p.m. of NO_2 for two to three hours, a lethal exposure in some cases.

In the main experiments five hours' exposure under conditions A caused no fatalities. There was some damage both to the trachea and to the lungs. To assess the main toxic hazard under these conditions we must turn to the results of the subsidiary experiments, in which animals were exposed for seven hours or more. In general there was in those animals dying from exposure to the fumes under conditions A severe damage to the whole of the respiratory tract, including tracheitis and pulmonary oedema. This suggests that irritants of the acrolein type, and probably also the nitrogen dioxide which was present, played a part in causing the deaths and that under these conditions they both constituted toxic hazards.

In all cases NO was present in greater quantity than NO_2. The toxicity of NO is somewhat similar to that of carbon monoxide (0·25% is lethal in 12 min.) (Flury and Zernik, 1931). It combines (as does CO) with haemoglobin, and probably reinforced the action of the CO which was present. Its effects are quite different from those of NO_2, and it cannot be considered a major toxic constituent of the fumes.

The somnolent behaviour of the mice in these experiments was probably caused by organic bodies having anaesthetic properties; this action may in some measure protect the mice from the toxic gases.

Conclusion

The conclusion reached as the result of these experiments is that the toxic substances in diesel exhaust fumes and their relative importance vary according to the working conditions of the engine.

With the engine and fuel used it appears that the chief toxic substances are:—(1) Under light load, irritants of the type of acrolein and, to a less extent, nitrogen dioxide, (2) under moderate load, nitrogen dioxide (NO_2), (3) under light load with restricted air intake, carbon monoxide and to a less extent irritants.

General Remarks

Further Work.—It has been shown that under some conditions the irritancy and the toxicity of diesel fumes are due to entirely different factors. The determination of two of the main toxic factors—nitrogen dioxide and carbon monoxide—is quite possible, though in the former case far from easy. Identification, and still more determination, of the substance or substances responsible for eye irritation and tracheal damage seem to be very difficult. Determination of groups of substances, such as aldehydes, can give only a general idea of how much irritant might be present. Attempts to deduce the irritant or toxic action of the fumes from their measured composition are, therefore, very liable to error. Comparative measurements of lachrymation time, or subjective estimates of the relative irritancy of fumes under different running conditions, are easy to make. It is, therefore, highly desirable that anyone working on, for instance, the variation of composition of the fumes with running conditions, should at the same time test irritant and lachrymatory properties of the fumes, and if possible investigate their toxicity. Our experiments B and C show the great change in irritancy which can occur without marked change in the chemical findings.

In general it must be emphasized that as many measurements as possible of engine running conditions, fume composition, and irritancy should be made at the same time. Argument from one engine to another, from one worker's analysis to another's, or even from one day's performance by an engine to the next day's, is liable to fall into error.

Nitrogen dioxide is formed in large quantities in the petrol engine (Hanson and Egerton, 1937). There is, therefore, no reason to suppose that the toxicity of the fumes from the petrol engine, even if it were not masked by their much greater carbon monoxide content, would be less than that of diesel fumes; they might however have less effect on the trachea and bronchi than do diesel fumes. The main objection to diesel fumes seems to be their irritant nature.

Summary

Experiments have been carried out to ascertain what are the main toxic constituents of diesel fumes.

A single cylinder diesel engine was run under four different conditions: light load, moderate load, moderate load with a worn injector, and light load with the air intake restricted.

Mice, guinea-pigs, and rabbits were exposed to the undiluted fumes and at the same time determinations of a number of constituents of the fumes were carried out. Pathological investigations were carried out on the exposed animals. Animals were also exposed to acrolein vapour to assess its toxicity and mode of action.

The results of the analysis of the fumes were in broad agreement with those of other workers, except in regard to the sulphur oxides content; the latter was unexpectedly low.

Under light load the engine produced highly acrid fumes. Five hours' exposure caused no deaths, but seven hours' exposure did so. It is concluded that organic irritants (possibly aldehydes) and also nitrogen dioxide were the main toxic constituents.

Under moderate load, the engine produced fumes which were less acrid but more lethal, a high proportion of the animals dying during or shortly after exposure. Nitrogen dioxide is considered to have been the main toxic agent.

Replacement of a sound injector by one said to be " worn " made the fumes much less acrid and also less lethal. The lower toxicity is considered to be due to a lower nitrogen dioxide content.

When the air intake of the engine was restricted, very acrid fumes with much oily particulate matter were produced. This fume was lethal to all animals exposed for five hours. Carbon monoxide was the chief toxic constituent.

Under the running conditions used very little black smoke was present in the exhaust.

The procedure to be followed in future research is discussed.

We are indebted to Mr. W. George, of University College, Swansea, to L/Cpl. M. J. Farr, R.E., who assisted in the chemical analysis, and to Mr. M. A. P. Hogg, who determined the fuel characteristics.

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BY

F. M. TROISI

From the Medical Clinic, University of Bologna, Italy

(RECEIVED FOR PUBLICATION SEPTEMBER 11, 1956)

Three workers died from carbon dioxide poisoning in a partly underground silo which had been almost completely emptied of green forage. Two died at once, the third after five days without regaining consciousness.

Several similar cases are reported in the literature. Wrede (1936) described a case of carbon dioxide poisoning which occurred in a cellar through the escape of gas from a beer barrel. The patient, seen four minutes after he had been removed from the cellar, was unconscious but recovered in a few minutes. The clinical picture was one of narcosis followed by excitation and could easily have been mistaken for drunkenness or epilepsy.

Bernauer and Blume (1937) reported the cases of three Chinese employees working in the hold of a ship in Canton port who were poisoned by carbon dioxide produced by the fermentation of rice and soya. One of them died at once, the second recovered quickly, and the third succumbed after four days without regaining consciousness.

Taeger (1937) reported the case of a youth of 22, who collapsed in a silo containing green forage. When removed from the silo he was completely unconscious, the pupils dilated and reacting feebly to light, and Cheyne-Stokes respiration was present. After 20 hours he began to improve slowly. Acrocyanosis then appeared in the hands and feet which was still present two years after the incident and was considered by the author to be aetiologically connected with the attack of carbon dioxide poisoning.

Symanski (1939) described four workers who lost consciousness in a silo containing green forage but recovered. One had aphasia for a short time after recovering consciousness, and in one case seen a week after the incident there was marked acrocyanosis of the hands.

Case Report

On May 20, 1954, a youth of 18 and a boy of 12 descended into a silo on a small farm. The silo was constructed of cement, cylindrical in shape, 6 metres high and about 4·5 metres in diameter, about half its height being buried in the ground, with only one opening in the roof, which was closed with a metallic cover. Green forage, mixed with beans, barley, and oats had been preserved in the silo, which had been nearly empty for about a month, with the cover in place. The two youths entered the silo to remove the remaining silage before replacing it with fresh material. Shortly after reaching the bottom of the silo they collapsed and became unconscious. A man of 61 standing nearby went into the silo to help them and he also lost consciousness. All three were dragged out with grappling hooks and artificial respiration was applied. The two youths were already dead. The elderly man was immediately taken to the nearest clinic. He appeared to be *in extremis*; the only sign of life was intermittent cardiac contractions. Artificial respiration was started at once and cardiac and respiratory stimulants were given together with 500 ml. plasma intravenously. After about an hour a few spontaneous respirations began and progressively in about 10 minutes tachypnoea developed. Consciousness was not regained at any time.

After five hours he was admitted to hospital completely unconscious, the superficial and deep reflexes, pupillary and corneal reflexes absent, a positive Babinski sign, more marked on the left side, double incontinence, tachycardia (120/min.) and tachypnoea (blood pressure 70/55 mm. Hg), and a temperature of 97·9° F. Lobeline and oxygen were administered, and a transfusion of 300 ml. of blood was given, preceded by bleeding; antibiotics were given prophylactically. In the first 15 hours the tachypnoea ceased but violent generalized convulsions recurred at half-hourly intervals. Four transfusions of 300 ml. of blood, preceded by bleeding, were given during the ensuing 24 hours and the blood pressure increased to 120/75 mm. Hg but fell again after a short time. The temperature rose to 100·4° F. Oxygen and antibiotics were given. Towards the end of the second and the beginning of the third day a partial but short-lived recovery of the abdominal and lower limb reflexes took place. On the afternoon of the second day the blood was examined chemically and spectroscopically; no carbon monoxide was present. On the third day the condition remained unchanged (blood pressure 85/60 mm.

Hg, temperature 101·3 F.) and a blood transfusion was given without previous bleeding as the pulse pressure was very low, together with oxygen and antibiotics. On the fourth day the clinical picture was similar (blood pressure 90/75 mm. Hg, temperature 102·2° F.), and treatment was given as on the previous day. On the fifth day, May 25, the temperature rose to 102·6° F. and finally to about 104° F. just before death. Throughout the illness there was complete loss of consciousness; all urine tests showed albumin; the non-protein nitrogen level in the blood in the first 24 hours was 52 mg. %, rising to 75 mg. % on the fourth day. From his friends it was ascertained that the patient had previously enjoyed good health and they could not remember any illnesses from which he had suffered.

The clinical picture was that of asphyxia, which did not cause death immediately, either because the length of time during which the patient remained in an atmosphere of complete anoxia was very short (less than eight minutes, a duration which is generally admitted to be quickly fatal), or perhaps because there was a reduced partial pressure of oxygen. This was a case of delayed death due to carbon dioxide intoxication and perhaps to anoxaemia also. The clinical picture was characteristic of prolonged partial anoxaemia such as occurs in cases of slowly induced asphyxia or under anaesthesia with nitrous oxide and less than 10% oxygen. In these conditions the central nervous system may be seriously damaged, and death may occur after hours or days during which respiration with pure air or oxygen proves ineffective (Henderson and Haggard, 1943).

The progressive rise in temperature, apparently not caused by any inflammatory condition, is worth noting, as similar increases in temperature were observed in the cases reported by Parade (1930) and by Bernauer and Blume (1937), which they regarded as being due to toxic effects on the thermoregulatory centre. In the incident now described, the cause of the fatal toxic effect in the three employees was evidently the carbon dioxide produced by fermentation of forage remaining in the silo, activated by the higher spring temperatures and accumulating on account of its higher density in the lower part of the closed silo. Chemical examination of a sample of forage taken from the silo revealed active fermentation; it was thought that weed killers and insecticides used in the fields might have left a residue which could have given rise to arsine or phosphine, but arsenic and phosphorus were absent from the sample. It is well known that in such conditions a high concentration of carbon dioxide may accumulate at the bottom of silos. Fermentation is determined by the nature and state of the forage (type, chemical composition, humidity, and enzyme complexes), by conditions of complete and incomplete anaerobiosis, by the initial and continuing temperatures, and by the type and amount of bacteria. Fermentation also results in the production of small quantities of hydrogen and methane.

The risk of carbon dioxide poisoning in silos, granaries, and holds of ships, where forage or grain is stored, has been known since ancient times and fatal cases continue to be reported from time to time in the daily press. Ramazzini (1713) warned that

> "a fact worthy of marvel is that from grain which has been long conserved in a closed chamber, for example in underground places as is the custom in Tuscany, arises an exhalation so dangerous as to be sufficient to cause death to anyone who enters such a place to collect the grain, unless the pernicious air is first allowed to escape for a while."

It is important to remember that a very considerable accumulation of carbon dioxide may take place in silos, whether they contain forage or grain. Apart from fermentation and putrefactive processes, an increased natural respiration of the grain readily occurs as may be deduced from the following data. One kilogram of grain containing 14-15% of moisture at a temperature of 18° C. produces 1·4mg., at 30° C. 7·5 mg., and at 52° C. 249 mg. of carbon dioxide in 24 hours. Again, 1 kg. of grain at a temperature of 18° C. produces, with a moisture content of 14 to 15% 1·4 mg., of 16·9% 123 mg., and of 33% 2,000 mg. of carbon dioxide in 24 hours (Bordewieck, 1936).

The type of silo described above, half-buried in the ground and with a single opening in the roof, should be prohibited; in addition all silos should be provided with a free opening at the base. Underground tanks and vats must be actively ventilated before they are entered, and the person entering such a chamber must be secured by a rope and be observed from outside. When it is necessary to enter a chamber where the presence of carbon dioxide is known or suspected, it is essential to use breathing apparatus which draws air from outside the chamber, or self-contained breathing apparatus. Other preventive measures to be adopted depend on the conditions under which the work is undertaken; it is important to give adequate instruction to the employees on the nature of the danger and its prevention. Posters may be displayed at farms indicating the dangers and the necessary preventive measures.

Methods used for testing enclosed atmospheres for the presence of carbon dioxide and of sufficient oxygen are to lower lighted candles or small caged animals into the chamber, and these are fairly reliable rough tests of safety. Parade (1930), however, besides noting differences in individual susceptibility to carbon dioxide poisoning, warned that

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warm-blooded experimental animals may only become unconscious at an atmospheric concentration of carbon dioxide as high as 25 to 30% and that it is therefore not sufficient to place rats, mice, or similar animals in small cages in the suspected atmosphere. In dogs, cats, rats, and mice a concentration of 10% of carbon dioxide in inspired air may have no observable effect but in man a similar concentration may be dangerous to life (Symanski, 1939). For this reason animals are reliable indicators only in a medium in which the partial pressure of oxygen is much reduced or absent. The candle test was indicated by Pliny the Elder, who says "Ergo vini faeci tanta vis est ut descendentes in cupas enecet. Experimentum demissa praebet lucerna, quamdiu extinguatur, periculum denuntians", thus recommending, as a precaution against fatal poisoning due to entering a wine vat, the lowering of a lighted candle into the vat and ascertaining the danger from the extinction of the flame. This type of test may be used quantitatively, as, at a concentration of 2% of carbon dioxide a candle flame assumes a reddish light, at 8 to 10% candles and oil lamps are extinguished, and at 12 to 14% alcohol lamps are extinguished but acetylene lamps are not extinguished until a concentration of 30 to 31% is reached (Holstein, 1949). There are, of course, numerous ways of estimating by physical or chemical methods the concentration of carbon dioxide in a working atmosphere.

Summary

Three fatal cases of carbon dioxide poisoning occurred in a half-underground silo nearly emptied of green forage.

One case, in which death occurred five days after the gassing incident, is described in detail.

The toxicology of carbon dioxide and its estimation in working environments are discussed and preventive measures described.

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OBITUARY

HUBERT WYERS

Dr. Hubert Wyers died at Rush Green Hospital, Romford, Essex, on December 12, 1956. By his untimely passing, at the comparatively early age of 56, industrial medicine has lost a fine physician and a devoted protagonist.

On leaving school he followed his father's occupation, engineering. It was not until his early twenties that he entered the Faculty of Arts at Edinburgh University where he read classics and philosophy. These studies influenced his whole life and were reflected in all his writings and lectures. On the removal of the family to the west of Scotland he transferred to the University of Glasgow. Here he graduated in Arts in 1927 and in Medicine in 1929. Following house appointments at hospitals in Glasgow he became an assistant in general practice in London, and from there he settled in general practice in Essex. He quickly became a very busy and highly esteemed family doctor. In due course he undertook part-time visiting appointments at several local factories. Following an operation for a perforated gastric ulcer May and Baker Limited offered him the opportunity to give more of his time to the medical service at their factory in Dagenham. Ultimately he decided to give up general practice so that he could devote his whole time to medicine in industry. His principal appointments were with May and Baker Limited, Murex, and the Cape Asbestos Company Limited.

Thus in his work he was confronted by the numerous and varied medical hazards of the fine chemicals, metallurgical, and asbestos industries. To all the emerging problems he brought a wide and mature experience, which was enhanced by careful observation, accurate recording, and shrewd judgment. He made important contributions to knowledge of poisoning by vanadium, cadmium, and methyl bromide. Skin diseases in industry were his special interest and until his death he was a clinical assistant in the Skin Department at Oldchurch Hospital. In 1947 he was awarded the degree of M.D. by Glasgow University with commendation for his thesis on " Asbestosis " and in 1948 he obtained the Diploma in Industrial Health of the Conjoint Examining Board in England, for which diploma he was later appointed examiner.

He was an enthusiastic member of the Association of Industrial Medical Officers, serving as member of Council and chairman of the London Group. He was editor of the *Transactions* of the Association from the formation of that journal in 1951 until his death. An honour which gave him particular satisfaction was his election as chairman of the South Essex division of the British Medical Association. He was one of the original four founder members of the Thackrah Club.

Despite his many appointments and other professional activities he found time to be a skilled beekeeper, including the preparation of mead, a keen gardener, and an expert collector of early Roman coins. And all through his life he never relaxed his interest in the classics and philosophy. Perhaps it was these studies which, about two years before his death, influenced him to become a convert to Roman Catholicism. In the Church and its priests he discovered deep kinship and consolation.

Hubert Wyers was a wise physician with a deep reverence for life. With Terence he could sincerely affirm: "homo sum, humani nil a me alienum puto." This was his concept of the physician in industry:

> " He should be a man of wide culture, versed in history and customs of the race and in the laws of thought. Above all, he should have a flame burning in his heart for the sick man. To use a phrase of G. K. Chesterton, he should not be able to see the mob for the men."

He was no ascetic; for him there was no greater pleasure than the company of good companions enjoying fine English food graced by what he would call " the old Falernian ". We shall remember him in the words of Horace: " integer vitae, scelerisque purus."

He is survived by his wife and his mother.

A. MEIKLEJOHN

MISCELLANEA

Association of Industrial Medical Officers: 21st Anniversary Meeting

The Association of Industrial Medical Officers celebrated the twenty-first anniversary of its foundation by holding an Anniversary Meeting in London from September 24 to 28, 1956. The scientific sessions of the meeting were held at the London School of Hygiene and Tropical Medicine. The papers and subsequent discussions will be fully reported in the *Transactions of the Association of Industrial Medical Officers.*

At a reception given by the London Group, the President of the Association, Dr. R. S. F. Schilling, was invested by Sir Wilson Jameson with the new jewel. The central feature of the presidential jewel is a portrait, in enamel and gold, of Charles Turner Thackrah. Past presidents of the Association were presented with miniature replicas of the jewel. Sir Wilson Jameson spoke of the early days of the Association and recalled that he had encouraged its formation when he was consulted by a small group of industrial medical officers. He had advised them to avoid medical politics and to develop on a professional and social basis. He believed that the success of the Association had resulted from the adoption of this policy.

The inaugural meeting was opened by Mr. Iain MacLeod, Minister of Labour and National Service. The Minister referred to the great progress which had been made in the development of industrial medicine, supported by the growth of the Association and by the many doctors who were serving in industry on a part-time or whole-time basis. Like his predecessors, he was actively interested in furthering these developments. Several members of the Association were also members of his recently appointed Industrial Health Advisory Committee. He had initiated two pilot surveys of industrial health, a general survey in Halifax and the other concerned with the pottery industry and centred on Stoke-on-Trent. In his Presidential Address, Dr. Schilling outlined the development and activities of the Association and referred to the recent publication by the Association of a booklet on the functions of an occupational health service.* He hoped that this booklet would be found useful both by managements and by doctors newly entering the field of occupational health.

Professor Sven Forssman described his experience of the health problems of the older workers in Swedish industry. He had undertaken a study in eight industries with 20,000 employees. Regular examinations were important, and were becoming increasingly so as the number of older people in the population increased. This social development was characteristic of countries with a high living standard. The increase of mechanization in industry was reducing the number of jobs requiring physical strength and might be expected to benefit the older workers, but they found it difficult to work at high speeds. There appeared to be two main groups of elderly workers: those who are in good health and those who had survived such illnesses as tuberculosis and diabetes because of improvements in medical treatment. Professor Forssman found that most employees over the usual retirement ages wished to continue at work. This was unrelated to pension rights and applied to both manual and clerical workers. Diseases of the circulatory system and of the organs of movements were the main causes of impaired health and working capacity of older workers. As has been found in other studies, older employees experienced more sickness absence than younger workers, particularly those employed on light work. The productivity of the older worker compared favourably with that of the average of workers as a whole. It was a mistake to put young and elderly employees to work together, because of their different speeds of work. Older workers should have longer midday breaks and rest pauses. The age of retirement should not be fixed but should be flexible and according to individual capacities.

The Mackenzie Industrial Health Lecture, sponsored by the British Medical Association, was delivered by Dr. R. S. F. Schilling. He said that the industrial medical officer was nowadays called upon to assess many physical and environmental factors concerning workpeople, and not merely to confine himself to the study of industrial disease. The methods of epidemiology were of much value in measuring hazards by comparing different occupational groups. Dr. Schilling referred to the work undertaken by Greenhow almost a century ago on mortality and morbidity rates. Recent research on coronary artery disease had shown that workers in physically active occupations experienced relatively less coronary disease than sedentary workers. An excess mortality of strippers and grinders in the cotton industry from cardiovascular-renal disease had been demonstrated. Statistics of sickness absence were essential for the study of morbidity but their use required considerable care, for they could be misleading as absence recorded as due to sickness did not always depend on pathology. Field surveys were of the greatest importance in the study of industrial disease, but their success depended on the cooperation of the population concerned. Observer errors in clinical and radiological signs were also very important.

* The Functions of an Occupational Health Service in an Industrial Organization, published by the Association of Industrial Medical Officers, London, 1956.

At a symposium-discussion on " Research in Occupational Health " the chair was taken by Dr. M. W. Goldblatt. The Earl of Verulam suggested that occupations might be classified as sedentary, peripatetic, peculiar, asymmetrical, or mobile. More research was needed into problems of seating and posture at work. Industry was not opposed to research, or to the application of knowledge acquired by research. There was likely to be a growing demand for steadily rising standards of working conditions. Sir Harold Himsworth, speaking on " The Relating of Research to Practice ", said that the separation between fundamental and applied research was essentially artificial. Progress depended on the smooth flow of ideas from one to the other. Unfortunately, workers at these two extremes tended to be widely apart in their ideas. There was need for a continuous chain of men with overlapping skills and experience, from the laboratory to the field. It was inadequate simply to mix men with the two different skills. Sir Alfred Roberts stressed the need for a comprehensive occupational health service which underlay all aspects of occupational health, including research. The essential requirements were a coordinated Government policy and a substantial increase in the resources devoted to occupational health. In providing additional resources consideration should be given to the establishment of an industrial hygiene service to assess environmental conditions and tackle the problem of occupational health risks arising from the introduction of new materials and processes. It was essential that field investigations should occupy a large part of any programme of occupational health research.

Dr. Katharine Williams read a paper on " The Impact on Medicine and Industry of the Use of Atomic Energy ". The demands of nuclear engineering had resulted in the development of new materials; high temperatures and intense radiations called for research and development in metallurgy. Radiations from reactors and isotopes could alter the physical properties of materials such as polythene. Radiation-induced sterilization could be used for the preservation of foodstuffs and for pharmaceutical and medical supplies.

The film " Calder Hall " was shown by Dr. Williams during this lecture.

A lecture-demonstration on " Weight-Handling " was given by Mr. R. J. McD. Maxwell. After an introductory talk on " cumulative strain " (as opposed to traumatic strain) Mr. Maxwell described six key factors in the technique of kinetic handling, *viz.*, proper hold, proper foot positions, straight back, arms into sides, chin in, and counterbalance. A demonstration of the different movements involved in handling boxes, bags, barrels, and other objects followed.

Professor R. E. Lane delivered the John C. Bridge Memorial Lecture on " The Place of Occupational Medicine in a Teaching Hospital ". He suggested that an occupational medicine unit improved the hospital service to patients, broadened medical teaching, and contributed to clinical research. Professor Lane described the work undertaken by his department at Manchester Royal Infirmary during the three-year period 1953-55. There were 511 new male patients, 177 of whom were suffering from chronic respiratory disease (including pneumoconiosis and byssinosis) and 44 were suffering from other industrial diseases. The lecture was illustrated with nine case histories, demonstrating the need for the hospital physician to gain a knowledge of the working conditions and social and economic circumstances of his patients. The resettlement of 180 men in industry was described; 18 months after resettlement 47% of these men remained settled in suitable work, 23% were not working and 28% could not be traced. The place of occupational health in undergraduate medical teaching was discussed. Clinical teachers should show the student how work can heal as well as harm; too often it is only the harmful effects of work that receive mention. Direct teaching should be undertaken at the occupational medicine clinic, where medico-social problems, including the patient's occupation, are dealt with as part of the whole clinical problem. Professor Lane emphasized the contribution to clinical research which could be made by the department of occupational medicine, quoting the work undertaken in his department on byssinosis and poisoning by cadmium fumes.

At the session on " Mental Health in Industry " the chair was taken by Dr. L. G. Norman. Dr. R. F. Tredgold spoke on " The Use of Psychiatry in Management Training Problems ". He said that there had been a great increase in formal management training programmes, most of which emphasized the need to understand the individual worker. Hence many firms incorporated teaching in psychiatry, either openly or camouflaged, in such programmes. Dr. Tredgold outlined the techniques which could best be used by the psychiatrist when taking part in management training and emphasized the need for the psychiatrist to be in close touch with the working environment and with the latest research. Dr. Alastair Heron read a paper on " Psychological Research in Industry " in which he perceived the rôle of the psychologist as that of an enquirer whose relatively objective findings might be found useful by such potential consumers as the industrial manager and his medical and personnel advisers. His enquiries should concern persons as individuals: the relations between persons; and those between persons and machines. The research psychologist should devote considerable attention to methods of training and he should always study employee satisfaction as well as individual efficiency. The adaptation of the marginal employee would always constitute a major challenge to the research psychologist in the occupational field. A paper on " Work, Payment, and Capacity " was given by Dr. Elliott Jaques, in which results were presented to show that level of work, or level of responsibility in jobs could be accurately measured. Measurement was based on the assessment of the maximum span of time during which an employee was expected to exercise discretion on his own account without review. This time-span measure could be applied to any job from shop floor to top executive level. Peace of mind for the individual in his work required that he be allocated a level of work that was consistent with his capacity and which kept pace with his growth in capacity; and that he be paid at a level consistent with the current time-span payment scale. Deviations from a consistent relationship between the individual's work, payment, and capacity

produced behavioural symptoms which could be predicted. Examples of such patterns of behaviour were described.

During the week visits were paid to a number of industrial organizations in the London area and there was a full social programme for members and their wives.

An exhibition of the medical services of the Ministry of Supply was open during the week at the Pillar Hall in the new Government buildings, Whitehall. Exhibits and illustrations were shown, covering many types of hazards and the protection required. Of particular interest was the resuscitation section in which the historical development of resuscitation procedures was illustrated with examples of old and modern types of equipment.

The anniversary dinner was held at the Royal College of Surgeons on the anniversary date, September 27, of the foundation of the Association 21 years before. The toast of the Association was proposed by Sir Harry Platt, who spoke of the need for all branches of medicine to work together. The President of the Association, Dr. R. S. F. Schilling, in his reply, foresaw a steadily developing future for occupational health services, bringing great benefit to industry. In proposing the toast of " Medicine in Industry " the Earl of Verulam recalled the many benefits to industrial management which an occupational health service provided. The toast of the guests was proposed by Dr. L. G. Norman, and Dame Florence Hancock replied.

Erratum

We much regret that the legends to the paper by J. N. Morris in the last number (**13**, 287) were omitted. They are as follows:—

FIG. 1.—Mortality in the " social classes " England and Wales, 1950. Males aged 20-64 inclusive.

FIG. 2.—Mortality from coronary heart disease England and Wales, 1950. Males aged 20-64 years inclusive.

Industrial Medicine and Hygiene

A Review by
L. G. NORMAN

This is the only book in the English language which brings together a series of authoritative essays comprehensively covering almost the whole field of industrial medicine and hygiene. Messrs. Butterworth & Company are to be congratulated on their foresight and enthusiasm, no doubt stimulated by the late Lord Horder, in developing this work. Dr. E. R. A. Merewether, as Editor, has not only achieved a personal masterpiece; he has placed British industrial medicine firmly in the forefront. To collate the balanced contributions of 33 individualistic experts, to make them speak roughly the same language, and avoid overlapping each other's contributions to any serious extent is a remarkable achievement. It has been a task which few would care to undertake and the three present volumes will remain a lasting tribute. There is indeed a galaxy of authors, each at the head of the field about which he writes. The work is perhaps least strong on the clinical side, but it does not set out to be a clinical text-book, and about half the authors, leaders in their own field, are not medical men. There are one or two omissions, which, together with a summary of recent advances, will, it is hoped, form the basis of a further volume or volumes in order that the book as a whole may be kept up to date. For example, there is little about the treatment of gassing casualties, human-engineering aspects of machine design, the clinical aspects of toxicology, accident prevention, the effects of shift work, and psychiatry in industry.

There is a good index, separate for each volume. The production is of a high standard throughout, on good quality art paper, with excellent illustrations, and with a refreshing absence of typographical errors.

Volume I

This volume was published in 1954 and is inevitably out of date in minor respects. It is interesting to note that progress seems to have been most marked in legislation. For example, the Mines and Quarries Act and the Food and Drugs Act, 1955 (and Regulations), have been passed since the relevant chapters in this volume were written.

The volume opens formidably but perhaps appropriately with an account of the work of the Factory Inspectorate by H.M. Chief Inspector of Factories. Sir George Barnett's brief and impeccable first chapter describes the history of the Factory Inspectorate and gives an account of its present-day activities. As this is

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Industrial Medicine and Hygiene. Vols. 1-3. Edited by E. R. A. Merewether. (Pp. ix + 426, vi + 451, vii + 617 respectively; illustrated. 70s. per volume; 10 gns. per set.) London, Butterworth Medical Publications: 1956.

a work on industrial medicine one had hoped to find a more complete account of the work of the medical inspectors. The reference to policy in regard to the assignment of "the medical side" to another ministry or department might have been better omitted. Mr. McCullough, a Deputy Chief Inspector of Factories, follows with a chapter on factory legislation which, as he says, is written not for the expert but for the man in the street. The doctor newly undertaking an industrial appointment will, however, find this chapter useful. The sections on heating, ventilation, and lighting go beyond legislative requirements, but this chapter may prove a helpful guide on what is acceptable to the Factory Inspectorate. An excellent description, somewhat beyond the requirements of hygiene, is given of the fencing of machinery. There is a useful list of the legislation covering factory owners and occupiers on the one hand and workpeople on the other. This chapter will also be found useful for reference, for example, on requirements covering young persons, and legislation concerning weight-lifting.

Dr. Reid follows with a masterly account of the application of statistical and epidemiological methods. In a mixture of statistics and common sense he leads us gently by the hand along difficult paths until the reader finds himself enthusiastically calculating square roots and standard deviations. Dr. Reid is rather optimistic in supposing that many factory managements keep adequate records of staff, adequate, that is, for the purpose of statistical investigations. This chapter might have been improved by the inclusion of actual examples of the practical use of morbidity and mortality statistics. There is perhaps some confusion between the personal sickness record of the individual employee, which is useful clinically, and the grouped records of numbers of employees which are of epidemiological use. The Industrial Health Research Board Report No. 85 is now out of date and should not be made the basis of sickness absence recording. Since this chapter was written the Registrar-General has published his recommendations concerning terminology in sickness absence recording and it is desirable that these terms should be generally adopted. In this work there is sometimes thought to be a danger of too large a statistical pyramid being built on the shifting sands of human data; Dr. Reid's sound common sense avoids this pitfall.

Dr. Amor describes the organization and operation of an industrial medical department, emphasizing throughout that the medical officer is part of the management team. Not all will agree that the medical officer should be responsible to the personnel manager. There is a practical account of the working environment, divided into physical, chemical, biological, and psychological environments. Descriptions are given, with a plan and photographs, of the siting and design of a medical department for both large and small organizations. These will be found useful by those who require to set up a new department but the author is against the provision of physiotherapy, for which accommodation is therefore not included. Useful examples are given of medical examination and report forms; the difficult question of disclosure to the employer of such information as diagnosis is not discussed. Dr. Amor has put much of his wisdom and experience into this chapter, which will be read with profit.

The organization of industrial and occupational nursing services is described by Miss Mann, formerly Industrial Nursing Organizer of the Royal College of Nursing, and it may be assumed that this chapter is in line with the general policy of the Royal College. Miss Mann does well to remind us of the report of the Health of Munition Workers Committee (1918) which referred to the industrial medical service as "this new preventive medicine which has as its object the removal of the occasion of disease and physical inefficiency combined with the husbanding of physical resources of the worker in such a way and to such a degree that he can exert his full powers unhampered, and with benefit to himself and all concerned". An excellent blueprint for today. Surprisingly Miss Mann does not emphasize the importance of the State Registration qualification for nurses who take charge of works surgeries. The sections on siting and accommodation of "health departments" somewhat overlap the previous chapter and much of the detailed lists of equipment could have been omitted. In the design of a new health department the provision of a combined consulting room (for consultations with the nurse) and nursing staff office seems inadequate and the accommodation listed for the department as a whole is incomplete. The standards of staffing recommended are of interest, and the references to rates of pay and conditions of service are in accordance with the recommendations of the Royal College of Nursing which are revised from time to time. The author does not indicate that the nurse's duties should take her outside the treatment room into the factory. The important question of administration of morphia by factory nurses is not discussed. Miss Mann considers that nursing staff should work to "standing orders", of which a copy should be posted in the treatment room; many medical officers, however, prefer to allow their nurse colleagues professional discretion in accordance with their ability.

Dr. N. Langdon Lloyd describes the Ministry of Supply Medical Service, and gives a valuable account of the work of a large industrial medical service. Examples are given of pre-employment medical examination forms and of other useful forms and certificates. No account is given of the levels of sickness absence experienced in royal ordnance factories following the introduction of a sick pay scheme, a cautionary tale which might have been a help to others. This excellent description of the work of a large industrial medical department will repay careful study by those who work in or join such a department. Of particular merit is the final section "*Esprit de Corps*" which should inculcate a strong sense of pride in belonging to a service with a high reputation.

The medical aspects of coal-mining are described by Dr. S. W. Fisher. A compact and useful account is given of history and legislation, not readily available elsewhere. There is a full account of miner's nystagmus, but no mention of Browne's work on kittens which supported the view that nystagmus was mainly due to

inadequate lighting. There is a useful account of underground operations. The " beat " diseases are adequately described in accordance with the Prescribed Diseases Regulations, although in fact there are cases which do not always seem to fit the Regulations, such as " beat shoulder ". Dermatitis is rather briefly described. There is an excellent account of gases in coal-mines (though not of dealing with gassing casualties) and of high temperatures and ventilation in mines. The extensive medical services now provided by the National Coal Board receive little description, perhaps because they fall under a different administration from that with which the author was associated.

Dr. Patricia Shaw writes on medical supervision in shops, warehouses, and offices. This is of particular interest in view of the promised Gowers legislation. A sympathetic and practical account is given of the nature of shop work, and of the special skills required. There are few health hazards for workers in shops, but Dr. Shaw rightly draws attention to the potential dangers of *x*-radiation in shoe fitting and of the dissemination of tuberculosis. No reference is, however, made to Alice Stewart's work on the spread of tuberculosis in large and small workrooms. Dr. Shaw is perhaps unduly restrictive in the selection of workers for shops and offices. It would have been interesting to see Dr. Shaw's views on the important question of large versus small offices; from the points of view of ventilation, distraction, and infection the small office seems to have considerable advantages over the large one. Staff accommodation is rather inadequately described. There is a helpful account of methods of medical supervision, particularly where there are multiple scattered units. The impression is left that this is a valuable account of medical services in shops and related premises which might, however, have been expanded to give a more complete account of legislation, environmental working conditions, and staff accommodation.

Dr. T. A. Lloyd-Davies contributes an essay on " The Young Worker " which is in quite a different style. It is a fine piece of writing, redolent with sympathy and keenly sensitive to the needs of older school children and young workers; a mixture of sound philosophy and common sense. The historical and social background of adolescents is analysed, drawing attention to the different social conditions after World Wars I and II. The table of contrast in " employment " conditions between school and work is of much interest. Clinical case reports of four " difficult " adolescents reveal the author's deep understanding; they are worthy of careful study and thought. Leisure interests, motivation in work, and after-school education are adequately discussed. This is a valuable essay on the social employment and medical problems of young adults which should be studied by all who are in any way responsible for the care of young people at work.

The responsibilities of the Ministry of Labour and National Service in regard to the disabled worker are considered by Mr. W. Taylor, a former Under-Secretary of the Ministry. It is refreshing to find a full and readable account of the working of the Disabled Persons (Employment) Act, 1946. Unemployment among registered disabled persons is considered and there is a detailed description of the work of the Disablement Resettlement Officer. In 1953, nearly 130,000 vacancies were filled from the Disabled Persons Register, which is a tribute to the administration of the Act. There still remain over 50,000 unemployed persons in Section I of the Disabled Live File (those who are regarded as capable of work under ordinary conditions), and this number should not be regarded complacently. The work of the Ministry's industrial rehabilitation units and government training centres is described in detail. The chapter concludes by describing the work of the Remploy factories and of voluntary organizations and local government authorities in assisting disabled persons. A subject which is a maze of confusion to many will be clarified by reading this chapter.

Dr. Donald Stewart appropriately contributes a chapter on rehabilitation, for he is medical adviser to the Austin Motor Company whose pioneer rehabilitation workshop is well known, and a member of the National Advisory Council on the Employment of the Disabled. Starting with rehabilitation in the hospital, progress has generally been disappointing since the Ministry of Health's Memorandum on Rehabilitation (1949), though the author might have mentioned a number of hospitals where the physical medicine specialist or orthopaedic surgeon has developed excellent rehabilitation services. The contribution of industry to rehabilitation is discussed, with a description of certain rehabilitation workshops. Many employers prefer to provide rehabilitation by means of alternative work, without segregating the employees concerned in separate workshops, and the value of this contribution to rehabilitation is perhaps not sufficiently emphasized. It is true that in this form of rehabilitation the patient is apt to be " lost " in the works, particularly if he fits his new environment well (a tribute to the medical officer), but it is surely the medical officer's responsibility to recall such cases for follow-up and so not lose sight of them. The influence of wage structure and incentive bonus schemes on rehabilitation arrangements, influences which may make or wreck a rehabilitation scheme, are not discussed. Special consideration is given to rehabilitation in pneumoconiosis, tuberculosis, heart desease, psychiatric cases, and the placing of blind persons. Many industrial medical officers doubt whether infectious persons suffering from tuberculosis should be placed in ordinary industry at all. In heart disease, Dr. Stewart gives a useful classification of functional activity, quoting the Work Capacity Report proposed by the American Heart Association in 1952. Dr. Stewart describes and generally criticizes the various schemes for matching human capacity against a job analysis. Industrial medical officers will usually agree with this view; most disabled cases have to be assessed individually and there is no substitute for the medical officer himself seeing and knowing the details of the job concerned. Useful appendices to this chapter list 20 sheltered workshops for the disabled (excluding Remploy factories and workshops for the blind) and describe a method of job analysis for resettlement purposes.

National Insurance benefits for sick and injured work-

people are described by Miss Mildred Riddelsdell, Under-Secretary of the Ministry of Pensions and National Insurance. A much too brief historical account of the origins of health insurance and workmen's compensation is followed by detailed descriptions of the current sickness benefit and industrial injury provisions. The historical reasons for the separation of these two schemes and the advantages and disadvantages of their amalgamation are unfortunately not discussed. Since this chapter was written there have been changes in the rates of benefit, various changes in the provisions for pneumoconiosis and byssinosis, and poisoning by cadmium fumes has been added to the list of prescribed diseases. The Beney Committee reported after this chapter was written and a summary of its findings would have provided a useful statement of present opinion concerning cover for occupational diseases. The chapter concludes with a list of the prescribed diseases and scheduled occupations.

Dr. Magnus Pyke contributes a chapter on " Feeding the Worker ". A full account of nutritional requirements for light and heavy workers is given. Unfortunately only one sample menu is included. Many industrial medical officers do not have sufficient concern with the nutritional values of canteen meals, but here will be found enough information in a brief space to answer any questions regarding the nutritional needs of employees. Dr. Pyke supports the administration of milk to lead-workers, although it is now regarded as being of no particular value in protecting workers against lead poisoning. Diets suitable for special categories of workers such as adolescents and night workers are described, but there is no mention of special dietary arrangements for medical cases such as those suffering from peptic ulcers and diabetes.

Volume I concludes with a chapter on canteen services by Miss D. Johnson, a Deputy Chief Inspector of Factories. A history of the development of works canteens is followed by descriptions of the siting, accommodation, equipment, management, and staffing of the canteen. From the industrial medical officer's point of view, it would have been useful to include a review of the principles of hygiene in canteens, together with some account of detailed hygienic requirements. The Food Hygiene Regulations (1955) were, of course, published after this contribution was written.

Volume II

This volume opens with a joint contribution from Dr. R. M. B. MacKenna and Dr. Sibyl Horner, a unique combination of a leading dermatologist and the Deputy Senior Medical Inspector of Factories who is the leading authority on administrative aspects of industrial dermatitis. The account of the legal aspects of occupational skin disease will be of much help to those who are concerned with this subject. The difficult question of compulsory notification of industrial dermatitis is adequately discussed. The failure, under present national insurance arrangements, to inform employers of cases of industrial dermatitis, which renders it impossible for employers to take action to prevent the disease, is mentioned, but it would have been preferable to read of the overcoming of this unfortunate obstacle. Skin diseases caused by occupation are discussed in sufficient detail for a work of this size, but there is of course ample material in this subject for the production of a separate volume. The summaries of the different conditions are, however, adequate and the photographs are excellent. Allergic contact dermatitis is briefly described and this section is followed by a somewhat lengthy discussion of cross-sensitization, with notes on the fundamental organic chemistry concerned. A good description of patch testing is given but the obvious failure of patch testing to detect sensitivity in advance of exposure is not mentioned. Skin cancer due to pitch and tar is well described, but a photograph of pitch warts might have been included. The uses, limitations, and composition of barrier creams are fully discussed and this section will be found most useful. Valuable hints on differential diagnosis of occupational skin conditions are given. There is some splitting of subjects, such as barrier creams, which appear in two places, and which might be brought together in a subsequent edition. This chapter is a well condensed summary of much information on occupational skin conditions.

Professor W. J. B. Riddell contributes a chapter on occupational ophthalmology. After the usual description of the anatomy and physiology of the eye (why is this always included in such articles?) there follow excellent accounts of many different types of injury to the eye and their treatment. Defective colour vision is briefly discussed; there is no reference to the invaluable report of the Physical Society on this subject. The Appendix gives the visual standards required by various civilian and military authorities, but it is not quite clear why lists of notifiable diseases have been included here.

Dr. C. B. Frisby contributes a chapter on occupational psychology. The Seven Point Plan, now well known, for the assessment of the individual is fully described. In the space available there is little detailed account of intelligence and aptitude tests, but the principles and uses of these tests are well set forth. The effects of faulty physical and psychological environments are reviewed, and there is a valuable account of the contribution which the industrial medical officer may make to relationships and morale, a role which gives him the opportunity to influence the philosophy of management.

Four separate authorities contribute chapters on compressed air, including one on medical aspects of compressed air illness by Professor W. D. M. Paton. The whole section of four chapters probably forms the best account of compressed air illness in the English language. The Appendix gives rules for compression and decompression, together with phase decompression tables.

Mr. H. W. Swann's excellent account of electrical accidents deals more with safety than health aspects, giving useful information about the prevention of electrical accidents and the safe handling of electrical equipment. The occurrence of hazards in various processes due to static electricity is described, together with methods of prevention.

Dr. H. E. Watts writes on explosives in industry, mainly from the point of view of those who are interested

inadequate lighting. There is a useful account of underground operations. The " beat " diseases are adequately described in accordance with the Prescribed Diseases Regulations, although in fact there are cases which do not always seem to fit the Regulations, such as " beat shoulder ". Dermatitis is rather briefly described. There is an excellent account of gases in coal-mines (though not of dealing with gassing casualties) and of high temperatures and ventilation in mines. The extensive medical services now provided by the National Coal Board receive little description, perhaps because they fall under a different administration from that with which the author was associated.

Dr. Patricia Shaw writes on medical supervision in shops, warehouses, and offices. This is of particular interest in view of the promised Gowers legislation. A sympathetic and practical account is given of the nature of shop work, and of the special skills required. There are few health hazards for workers in shops, but Dr. Shaw rightly draws attention to the potential dangers of *x*-radiation in shoe fitting and of the dissemination of tuberculosis. No reference is, however, made to Alice Stewart's work on the spread of tuberculosis in large and small workrooms. Dr. Shaw is perhaps unduly restrictive in the selection of workers for shops and offices. It would have been interesting to see Dr. Shaw's views on the important question of large versus small offices; from the points of view of ventilation, distraction, and infection the small office seems to have considerable advantages over the large one. Staff accommodation is rather inadequately described. There is a helpful account of methods of medical supervision, particularly where there are multiple scattered units. The impression is left that this is a valuable account of medical services in shops and related premises which might, however, have been expanded to give a more complete account of legislation, environmental working conditions, and staff accommodation.

Dr. T. A. Lloyd-Davies contributes an essay on " The Young Worker " which is in quite a different style. It is a fine piece of writing, redolent with sympathy and keenly sensitive to the needs of older school children and young workers; a mixture of sound philosophy and common sense. The historical and social background of adolescents is analysed, drawing attention to the different social conditions after World Wars I and II. The table of contrast in " employment " conditions between school and work is of much interest. Clinical case reports of four " difficult " adolescents reveal the author's deep understanding; they are worthy of careful study and thought. Leisure interests, motivation in work, and after-school education are adequately discussed. This is a valuable essay on the social employment and medical problems of young adults which should be studied by all who are in any way responsible for the care of young people at work.

The responsibilities of the Ministry of Labour and National Service in regard to the disabled worker are considered by Mr. W. Taylor, a former Under-Secretary of the Ministry. It is refreshing to find a full and readable account of the working of the Disabled Persons (Employment) Act, 1946. Unemployment among registered disabled persons is considered and there is a detailed description of the work of the Disablement Resettlement Officer. In 1953, nearly 130,000 vacancies were filled from the Disabled Persons Register, which is a tribute to the administration of the Act. There still remain over 50,000 unemployed persons in Section I of the Disabled Live File (those who are regarded as capable of work under ordinary conditions), and this number should not be regarded complacently. The work of the Ministry's industrial rehabilitation units and government training centres is described in detail. The chapter concludes by describing the work of the Remploy factories and of voluntary organizations and local government authorities in assisting disabled persons. A subject which is a maze of confusion to many will be clarified by reading this chapter.

Dr. Donald Stewart appropriately contributes a chapter on rehabilitation, for he is medical adviser to the Austin Motor Company whose pioneer rehabilitation workshop is well known, and a member of the National Advisory Council on the Employment of the Disabled. Starting with rehabilitation in the hospital, progress has generally been disappointing since the Ministry of Health's Memorandum on Rehabilitation (1949), though the author might have mentioned a number of hospitals where the physical medicine specialist or orthopaedic surgeon has developed excellent rehabilitation services. The contribution of industry to rehabilitation is discussed, with a description of certain rehabilitation workshops. Many employers prefer to provide rehabilitation by means of alternative work, without segregating the employees concerned in separate workshops, and the value of this contribution to rehabilitation is perhaps not sufficiently emphasized. It is true that in this form of rehabilitation the patient is apt to be " lost " in the works, particularly if he fits his new environment well (a tribute to the medical officer), but it is surely the medical officer's responsibility to recall such cases for follow-up and so not lose sight of them. The influence of wage structure and incentive bonus schemes on rehabilitation arrangements, influences which may make or wreck a rehabilitation scheme, are not discussed. Special consideration is given to rehabilitation in pneumoconiosis, tuberculosis, heart desease, psychiatric cases, and the placing of blind persons. Many industrial medical officers doubt whether infectious persons suffering from tuberculosis should be placed in ordinary industry at all. In heart disease, Dr. Stewart gives a useful classification of functional activity, quoting the Work Capacity Report proposed by the American Heart Association in 1952. Dr. Stewart describes and generally criticizes the various schemes for matching human capacity against a job analysis. Industrial medical officers will usually agree with this view; most disabled cases have to be assessed individually and there is no substitute for the medical officer himself seeing and knowing the details of the job concerned. Useful appendices to this chapter list 20 sheltered workshops for the disabled (excluding Remploy factories and workshops for the blind) and describe a method of job analysis for resettlement purposes.

National Insurance benefits for sick and injured work-

people are described by Miss Mildred Riddelsdell, Under-Secretary of the Ministry of Pensions and National Insurance. A much too brief historical account of the origins of health insurance and workmen's compensation is followed by detailed descriptions of the current sickness benefit and industrial injury provisions. The historical reasons for the separation of these two schemes and the advantages and disadvantages of their amalgamation are unfortunately not discussed. Since this chapter was written there have been changes in the rates of benefit, various changes in the provisions for pneumoconiosis and byssinosis, and poisoning by cadmium fumes has been added to the list of prescribed diseases. The Beney Committee reported after this chapter was written and a summary of its findings would have provided a useful statement of present opinion concerning cover for occupational diseases. The chapter concludes with a list of the prescribed diseases and scheduled occupations.

Dr. Magnus Pyke contributes a chapter on " Feeding the Worker ". A full account of nutritional requirements for light and heavy workers is given. Unfortunately only one sample menu is included. Many industrial medical officers do not have sufficient concern with the nutritional values of canteen meals, but here will be found enough information in a brief space to answer any questions regarding the nutritional needs of employees. Dr. Pyke supports the administration of milk to lead-workers, although it is now regarded as being of no particular value in protecting workers against lead poisoning. Diets suitable for special categories of workers such as adolescents and night workers are described, but there is no mention of special dietary arrangements for medical cases such as those suffering from peptic ulcers and diabetes.

Volume I concludes with a chapter on canteen services by Miss D. Johnson, a Deputy Chief Inspector of Factories. A history of the development of works canteens is followed by descriptions of the siting, accommodation, equipment, management, and staffing of the canteen. From the industrial medical officer's point of view, it would have been useful to include a review of the principles of hygiene in canteens, together with some account of detailed hygienic requirements. The Food Hygiene Regulations (1955) were, of course, published after this contribution was written.

Volume II

This volume opens with a joint contribution from Dr. R. M. B. MacKenna and Dr. Sibyl Horner, a unique combination of a leading dermatologist and the Deputy Senior Medical Inspector of Factories who is the leading authority on administrative aspects of industrial dermatitis. The account of the legal aspects of occupational skin disease will be of much help to those who are concerned with this subject. The difficult question of compulsory notification of industrial dermatitis is adequately discussed. The failure, under present national insurance arrangements, to inform employers of cases of industrial dermatitis, which renders it impossible for employers to take action to prevent the disease, is mentioned, but it would have been preferable to read of the overcoming of this unfortunate obstacle. Skin diseases caused by occupation are discussed in sufficient detail for a work of this size, but there is of course ample material in this subject for the production of a separate volume. The summaries of the different conditions are, however, adequate and the photographs are excellent. Allergic contact dermatitis is briefly described and this section is followed by a somewhat lengthy discussion of cross-sensitization, with notes on the fundamental organic chemistry concerned. A good description of patch testing is given but the obvious failure of patch testing to detect sensitivity in advance of exposure is not mentioned. Skin cancer due to pitch and tar is well described, but a photograph of pitch warts might have been included. The uses, limitations, and composition of barrier creams are fully discussed and this section will be found most useful. Valuable hints on differential diagnosis of occupational skin conditions are given. There is some splitting of subjects, such as barrier creams, which appear in two places, and which might be brought together in a subsequent edition. This chapter is a well condensed summary of much information on occupational skin conditions.

Professor W. J. B. Riddell contributes a chapter on occupational ophthalmology. After the usual description of the anatomy and physiology of the eye (why is this always included in such articles?) there follow excellent accounts of many different types of injury to the eye and their treatment. Defective colour vision is briefly discussed; there is no reference to the invaluable report of the Physical Society on this subject. The Appendix gives the visual standards required by various civilian and military authorities, but it is not quite clear why lists of notifiable diseases have been included here.

Dr. C. B. Frisby contributes a chapter on occupational psychology. The Seven Point Plan, now well known, for the assessment of the individual is fully described. In the space available there is little detailed account of intelligence and aptitude tests, but the principles and uses of these tests are well set forth. The effects of faulty physical and psychological environments are reviewed, and there is a valuable account of the contribution which the industrial medical officer may make to relationships and morale, a role which gives him the opportunity to influence the philosophy of management.

Four separate authorities contribute chapters on compressed air, including one on medical aspects of compressed air illness by Professor W. D. M. Paton. The whole section of four chapters probably forms the best account of compressed air illness in the English language. The Appendix gives rules for compression and decompression, together with phase decompression tables.

Mr. H. W. Swann's excellent account of electrical accidents deals more with safety than health aspects, giving useful information about the prevention of electrical accidents and the safe handling of electrical equipment. The occurrence of hazards in various processes due to static electricity is described, together with methods of prevention.

Dr. H. E. Watts writes on explosives in industry, mainly from the point of view of those who are interested

in the manufacture and storage of explosives. Safety measures are fully discussed and there is an adequate account of legal requirements relating to the manufacture, sale, conveyance, and importation of explosives, but there is little of medical interest in this chapter.

Dr. Thomas Bedford's chapter on heating and ventilation, on the other hand, contains just what is required by the industrial medical officer. Three scales of warmth are described, with a detailed account of the ranges of thermal comfort. In addition to a full description of the theoretical requirements for satisfactory heating and ventilation, there is practical guidance on the best methods of heating and ventilating workrooms. Incidentally, in an investigation of winter comfort zones for sedentary workers, undertaken in 1936, variations due to observer error were eliminated by having only one observer, a method which would not now be wholly acceptable. This chapter is invaluable for reference.

Dr. H. C. Weston writes on " lighting, glare, and efficiency ". An account of theoretical requirements for artificial lighting is given, but there is little description of the different methods of installation. More detailed information about the type, number, spacing, mounting-height, and light-output of lighting units would have been useful. The standards of illumination for different visual tasks recommended by the leading authorities might also have been usefully included. There is a full account of glare and its prevention. The section on fluorescent lighting will be found useful in advising on installations and in dealing with complaints about this form of lighting.

" Intense Sound and Ultra-Sound " is the title of the chapter by Air Vice-Marshal E. D. Dalziel Dickson. This chapter is of particular interest as people seem to be developing increasing sensitivity to noise. Physical methods of evaluating and measuring noise are described and a full account is given of the effects to noise on hearing. Useful practical accounts are given of the methods of reducing excessive noise in workrooms, but curiously there is no mention of acoustic tiles, of double windows, or of siting opposing wall surfaces at an angle so as to reduce the reverberatory effects of reflection.

The biological and clinical effects of ionizing radiations are considered by Dr. J. F. Loutit. Miners' cancer of the bronchus, and occupational disease of radiologists, radium technicians, and dial painters are described. There is an interesting account of the clinical effects of radiation, divided into non-threshold effects, which are cumulative and from which there is no recovery, and threshold effects, from which there may be a considerable measure of recovery.

This volume concludes with a chapter on protection against ionizing radiations by Dr. W. Binks. It is a valuable supplement to the Factory Department publication on precautions in the use of ionizing radiations and goes into considerable detail in the theory of this subject. The theoretical physics in this chapter, invaluable to the specialist, is, however, not likely to be of interest to the average medical practitioner. Nevertheless, industrial medical officers should undoubtedly make this chapter the object of study in view of the increasing use of radioactive isotopes in industry.

Volume III

This volume appeared somewhat later than the first two, and was published in May, 1956. It was worth waiting for. Volume III enhances the standard of the whole work. Silicosis and other fibrotic pneumoconioses are described by Dr. Andrew Meiklejohn in a masterly chapter of 120 pages. Current experience, mainly in Great Britain, is presented in a general survey, with the emphasis on practical problems rather than on scientific detail. Existing knowledge is well summarized and, as all who know Dr. Meiklejohn will appreciate, he has not shirked the discussion of controversial issues where necessary. The historical survey is regrettably brief, but no doubt limited by the space available. The photomicrographs of pathological specimens are well reproduced, but the small photographs of radiographs necessarily lose definition and detail; they are usefully included for the sake of completeness but there is no substitute for the study of actual radiographs. The author indicates where specimen radiographs may be obtained for study. After a discussion on the meaning and interpretation of the term " pneumoconiosis " the definition is left in some confusion; this is certainly a statement of the present position and the author has put the different views forward fairly. Theories of the development of pneumoconiosis are briefly and adequately described. Practical hints on the conduct of pathological examinations are given in the section on morbid anatomy. The author appeals for a critical re-examination of the view that massive fibrosis is due to tuberculosis infection. " . . . it is not yet disproved that massive fibrosis can result from intensity of dust exposure alone . . . ". The section on diagnosis is particularly good; it is based on the author's personal experience. There is much sound advice here, for example: " Doctors should warn patients against patent medicines and nips of brandy, if only to safeguard their financial resources. There is no better remedy than work; it keeps the mind occupied and avoids the introspection and depression of idleness, in which the patient becomes querulous of everybody and everything." The value of radiology in diagnosis is clearly expressed. The use of standard films, as a means of reducing observer-error in diagnosis, is not considered to be of much value to experienced observers. The danger is expressed that periodic medical examinations may degenerate into a sterile routine and the author emphasizes that all such examinations should have a clearly defined purpose, a view which might well be applied in a much wider field than that of pneumoconiosis alone. There is a full account of aluminium treatment, and the social aspects and the assessment of disablement. Recent work on the assessment of cardio-respiratory function is, however, rather too briefly described. There is some repetition in the description of silicosis of material that was given under the general heading of pneumoconiosis, but the account of silicosis stands well by itself and is an excellent introduction to the subject for the interested practitioner. Coal-miners' pneumoconiosis and asbestosis are similarly well described. The whole chapter is well written and should become a standard work of lasting value.

Dr. A. T. Doig follows with a chapter on dust diseases,

excluding the fibrotic pneumoconioses. This is an interesting and readable account which adequately covers the known examples of these conditions. Siderosis, due to inhalation of iron dust or fume, is fully described in a section which will remain useful for reference on this subject. The differential diagnosis between siderosis and silicosis is well summarized and cases of mixed exposure are briefly described. Short descriptions of stannosis, barytosis, and the effects of inhalation of calcium compounds complete the survey of pneumoconiosis due to metallic dusts. Acute pneumonitis due to metallic dusts and fumes is described, but the accounts of these conditions might perhaps have been more complete in view of the increasing importance of this subject. For example, the effects of vanadium pentoxide on cleaners of oil-fired boilers are but briefly described and there is no reference to Williams' work on this subject. Poisoning by cadmium fume (but not dust) has been made a prescribed disease since this chapter was written. Chronic pneumonitis due to beryllium is well described and there is a rather brief account of occupational cancer of the respiratory tract. The chapter concludes with a section on diseases due to vegetable dust, in which byssinosis is well described, but there is no reference to Schilling's Milroy Lectures (1956) on this subject, which were perhaps delivered after this chapter was written. Brief references are made to other diseases associated with animal or vegetable dusts. This chapter is successful in setting out the present position of knowledge without going into great detail; it will be useful for study and reference.

Those who work in industrial medicine have known for some time that Dr. M. W. Goldblatt and his wife had been working on the chapter on industrial carcinogenesis and toxicology for this book. It is a monumental work of nearly 400 pages, which will for long remain a tribute to the authors' patient recording and summarizing of present knowledge on many aspects of their subject. To have summarized all aspects would have required a work of many volumes and the authors have selected those subjects most likely to be of practical importance to industrial physicians. Part I deals with occupational carcinogenesis and first discusses the effects of chromium, arsenic, and nickel. There is no reference to Bidstrup's most recent paper on chromates. Although a satisfactory experimental basis is lacking in the case of chromium and arsenic, the authors review comprehensively and thoroughly the evidence for the carcinogenetic activity of these elements and their compounds. There is an interesting discussion of possible biochemical reactions which may lead to tumour formation. Our present knowledge of nickel acting as a carcinogen is well summarized. There is an excellent section on asbestos as a carcinogen, which might, however, have been better included in the previous chapter under asbestosis; this section includes a good description of co-carcinogenesis. In the following section on aromatic amines the authors reveal the advantage of bringing together the two disciplines, organic chemistry and medicine. Starting with the relatively simple theoretical basis of the synthesis of magenta (fuchsine), rosaniline, and methyl violet, the authors go on to describe the chemical theory involved in the more complex dyestuffs and intermediates, such as safranine, congo red, and auramine, developed in the earlier years of this century. At each stage in the formulation of the chemical theory the authors refer to the position then reached concerning views on the causation of bladder tumours. This parallel development of chemical research and knowledge of tumours of the bladder will be of more interest to specialists than to general practitioners, although it is through research on these lines that further knowledge of the origin of bladder papillomata is likely to be acquired. The synthesis of the two naphthylamines is next described, with an account of the evidence for the carcinogenicity of β naphthylamine. A suggested method for eliminating β naphthylamine as an intermediate by isolating it only as a sulphonate, maintaining sulphonation by the use of sulphonated β naphthol, is described, but it is unlikely that some of the uses of the intermediate could be met in this way. Experience in Germany and Italy between the wars in the use of naphthylamines and other intermediates is well documented. The chemistry of benzidine manufacture is next described, with a summary of the convincing evidence that this substance is also carcinogenic. Recent advances in the diagnosis of cancer of the bladder are well described, and a full account is given of recent experimental work. In published results on carcinogenic azo-benzene derivatives, it is striking that the parent azo-benzene is non-carcinogenic, whereas only those which yield an ortho-methyl-amino derivative on reduction fission are carcinogenic. As a matter of practical guidance in considering the possible carcinogenicity of any amine, a blocked para position to the amino group would facilitate ortho hydroxylation and the formation of the carcinogen. The implications of this work are fundamental in foreseeing possible carcinogens in the organic chemical industry. The section on beryllium would have been better transferred to the previous chapter.

Under the comprehensive title " Soot " there is a short description of the history of " chimney-sweepers' disease " followed by an excellent account of pyrene derivatives as carcinogens. A full description of the origin and chemical causation of tar and pitch warts follows; there are no clinical descriptions in this section of the book.

The section concludes with a useful practical description of the carcinogenicity of mineral oils, which, on account of their ubiquity, should be studied by all industrial physicians; a list is given of recommendations for the protection of workers who come into contact with mineral oil.

Part II of the chapter deals with some aspects of industrial toxicology. The approach is original; each problem is considered first from the chemical and biochemical angle. Most previous works begin with the presentation of signs and symptoms resulting from the use of certain industrial products, and follow with the methods advocated for prevention and treatment. The authors, however, deal with the subject quite differently. For example, under the general heading " inhibitors of phosphorylation ", is included an account of the biochemistry of this process, followed by a description of the effect of dinitrophenols and chlorinated phenols and

including incidentally an excellent account of the toxicology of dinitro-ortho-cresol. Once the reader has mastered this different approach he will find that the authors have considered many aspects of industrial toxicology from the essential angle of their biochemical origin, an approach which must surely form the fundamental basis of this subject in future. At present there is just sufficient knowledge for a start to be made and the authors have carried out their difficult enterprise extremely well.

XIIth International Congress on Occupational Health

The XIIth International Congress on Occupational Health will be held in Helsinki from July 1 to 6, 1957.

The preliminary programme is divided into (1) Congress subjects, (2) section subjects, (3) reviews, (4) round table, and (5) group meetings. The Congress subjects are industrial noise, evaluation of invalidity, industrial hygiene norms, and cardiacs and work.

There will be an opportunity before and after the Congress for members to visit and acquaint themselves with the organization and operation of occupational medicine and hygiene in other Scandinavian countries. There will be an excursion on July 7 to industrial establishments and hospitals in various parts of Finland.

The official languages of the Congress are English, French, German and Spanish.

The Nicolo Castellino prize, value 3,000,000 lire, will be awarded for the first time at the Congress at Helsinki. The award will be made for what, in the opinion of the assessors, is adjudged to be the most outstanding investigation in the field of occupational health by a research worker, who must be under the age of 35 years and is not the holder of a university chair. Details of the competition will be published at an early date by the Directors of the Fund or can be obtained direct from Directors of Nicolo Castellino Prize, The University, Naples, Italy.

A film competition will be organized in connexion with the Congress.

The Congress fee for members is £5 8s. and for associate members £3 12s.

Persons who wish to present original scientific papers should state the title of the paper, together with a summary of not more than 200 words in English and French, and should reach the Committee not later than March 1, 1957.

The final detailed programme and application forms will be posted early in 1957. Notification of intention to be present must be made by May 1, 1957. Hotel accommodation cannot be guaranteed after that date.

Further information relating to the Congress may be had on application to the Organizing Committee, c/o Työterveyslaitos, Haartmaninkatu 1, Helsinki-Töölö. The Chairman is Dr. Leo Noro and the Secretary-General Dr. Pentti Sumari.

Percivall Pott (1714-1788) and Chimney Sweepers' Cancer of the Scrotum

JOHN R. BROWN and JOHN L. THORNTON

From the Department of Applied Physiology, London School of Hygiene and Tropical Medicine, and the Medical College Library, St. Bartholomew's Hospital, London

With the possible exception of John Hunter, Percivall Pott is remembered as the most eminent surgical writer of his period, and the premier position he held in his profession was recognized not only in England but abroad. The fact that he is associated eponymously with several conditions has ensured that his name is still constantly before the medical profession but the facts of his career are little appreciated. Percivall Pott has not been the subject of a full-scale biography, and there are certain divergencies of opinion among those who have written about him. However, the following brief facts outline the activities of one who published the first description of an occupational cancer, and who should be numbered among the pioneers of occupational medicine.

Born in 1714 in Threadneedle Street, London, on the present site of the Bank of England, Percivall Pott was educated with a view to his becoming a clergyman.

However, on August 1, 1729, he was apprenticed to Edward Nourse (1701-1761), surgeon at St. Bartholomew's Hospital, paying a premium of £210 for the seven years' apprenticeship. Nourse gave lectures at his house in Aldersgate Street, at Barber-Surgeon's Hall, and possibly at St. Bartholomew's Hospital, and Pott became his prosector. On September 7, 1736, Pott was awarded the Grand Diploma by the Court of Examiners of the Barber Surgeons Company. In 1739 he applied unsuccessfully for the vacant assistant-surgeoncy at St. Bartholomew's Hospital, but on March 7, 1744/5, was appointed to that office. On November 30, 1749, he became full surgeon to the Hospital, occupying that position until he resigned on July 12, 1787. He was then nominated a Governor.

When Percivall Pott's mother died in 1746 he married Sarah Cruttenden, the daughter of a Director of the East India Company, and they had five sons (one died in infancy) and four daughters, one of whom married Sir James Earle. One of his sons, Joseph Holden Pott, became Archdeacon of St. Albans and London, and a great-great-grandson became Archdeacon of Berkshire.

Pott lived successively in Fenchurch Street, Bow Lane, Watling Street, Lincolns Inn Fields, and Prince's Street, Hanover Square. In 1756, while riding down the Old Kent Road to visit the Lock Hospital he was thrown from his horse, whereby he suffered a compound fracture of the ankle. During his convalescence Pott started to write a treatise on ruptures, and it is possible that but for his accident Pott would not have turned to authorship, and surgical literature would have been the poorer. His writings went into numerous editions, and several were translated into foreign languages (Lloyd, 1933; Horder, 1894). *A treatise on ruptures* and *An account of a particular kind of rupture frequently attendant upon new-born children* were published in 1756. His other important books include *Observations on that disorder of the corner of the eye, commonly called fistula lachrymalis* (1758); *Observations on the nature and consequences of wounds and contusions of the head* (1760), which includes the first description of " Pott's puffy tumour ", a circumscribed oedema of the scalp associated with osteomyelitis of the skull; *Practical remarks upon the hydrocele or watery rupture* (1762); *Remarks on the disease, commonly called a fistula in ano* (1765); *Observations on the nature and consequences of those injuries to which the head is liable from external violence* (1768); *Some few general remarks upon fractures and dislocations* (1768), in which he described " Pott's fracture " (pp. 57-64), *Remarks on that kind of palsy of the lower limbs, which is frequently found to accompany a curvature of the spine* (1779), his classical description of spinal curvature due to tuberculous caries, although Pott did not recognize its tuberculous nature, which became known as " Pott's disease "; and *Further remarks on the useless state of the lower limbs, in consequence of a curvature of the spine* (1782).

Several editions of his collected writings were published, and his writings remain of interest today. Pott died on December 22, 1788, and his influence on surgery continued to be exerted by his many pupils, among whom were John Hunter and John Abernethy.

Percivall Pott's account of chimney sweepers' cancer has been cited as the first description of an occupational cancer. It was published in 1775 in his *Chirurgical observations Relative to the Cataract, the Polypus of the Nose, the Cancer of the Scrotum, [etc.]*, London. The following is the complete text of the essay.

Cancer Scroti

" Ramazzini (sic) has written a book de morbis artificum; the Colic of Poictou is a well-known distemper, and every body is acquainted with the disorders to which painters, plummers, glaziers, and the workers in white lead, are liable; but there is a disease as peculiar to a certain set of people, which has not, at least to my knowledge, been publickly noticed; I mean the chimney-sweepers' cancer.

It is a disease which always makes its first attack on, and its first appearance in, the inferior part of the scrotum; where it produces a superficial, painful, ragged, ill-looking sore, with hard and rising edges. The trade call it the soot-wart. I never saw it under the age of puberty, which is, I suppose, one reason, why it is generally taken, both by patient and surgeon, for venereal, and being treated with mercurials, is thereby soon and much exasperated: in no great length of time, it pervades the skin, dartos, and membranes of the scrotum, and seizes the testicle, which it inlarges, hardens, and renders truly and thoroughly distempered; from whence it makes its way up the spermatic process into the abdomen, most frequently indurating, and spoiling the inguinal glands: when arrived within the abdomen, it affects some of the viscera, and then very soon becomes painfully destructive.

The fate of these people seems singularly hard; in their early infancy, they are most frequently treated with great brutality, and almost starved with cold and hunger; they are thrust up narrow, and sometimes hot chimnies, where they are bruised, burned, and almost suffocated; and when they get to puberty, become peculiarly liable to a most noisome, painful, and fatal disease.

Of this last circumstance there is not the least doubt, though perhaps it may not have been sufficiently attended to, to make it generally known. Other people have cancers of the same parts; and so have others, besides lead-workers, the Poictou colic, and the consequent paralysis; but it is nevertheless a disease to which they are peculiarly liable; and so are chimney-sweepers to the cancer of the scrotum and testicles.

If there be any chance of putting a stop to, or preventing this mischief, it must be the immediate removal of the part affected; I mean that part of the scrotum where the sore is, for if it be suffered to remain until the virus has seized the testicle, it is generally too late even for castration. I have many times made the experiment; but though the sores, after such operation, have, in some instances, healed kindly, and the patients have gone from the hospital seemingly well, yet, in the space of a few months, it has generally happened, that they have returned either with the same disease in the other testicle, or in the glands of the groin, or with such wan complexions, such pale, leaden, countenances, such a total loss of strength, and such frequent and acute internal pains, as have sufficiently proved a diseased state of some of the viscera, and which have soon been followed by a painful death.

If extirpation ever bids fair for the cure of a cancer, it seems to be in this case; but then the operation should be immediate, and before the habit is tainted. The disease, in these people, seems to derive its origin from a lodgment of soot in the rugae of the scrotum, and at

first not to be a disease of the habit. In other cases of a cancerous nature, in which the habit is too frequently concerned, we have not often so fair a prospect of success by the removal of the distempered part; and are obliged to be content with means, which I wish I could say were truly palliative; but here the subjects are young, in general in good health, at least at first; the disease brought on them by their occupation, and in all probability local; which last circumstance may, I think, be fairly presumed from its always seizing the same part: all this makes it (at first) a very different case from a cancer which appears in an elderly man, whose fluids are become acrimonious from time, as well as other causes; or from the same kind of complaint in women who have ceased to menstruate. But be all this as it may, the scrotum is no vital organ, nor can the loss of a part of it ever be attended with any, the smallest degree of inconvenience; and if a life can be preserved by the removal of all that portion that is distempered, it will be a very good and easy composition; for when the disease has got head, it is rapid in its progress, painful in all its attacks, and most certainly destructive in its event."

Pott states that he had not seen a case under the age of puberty, but in the edition of *The Chirurgical works of Percivall Pott* edited by Sir James Earle, the latter added a footnote (Vol. 3, 1808, p. 178) mentioning that he had encountered a case in St. Bartholomew's Hospital, in a child of 8 who was an apprentice to a chimney sweeper. He had shown the case to Pott, who acknowledged it to be one of cancer of the scrotum. In a further annotation (*Ibid.*, pp. 180-183) Earle mentions that Pott appeared to believe that the disease was peculiar to chimney sweepers, despite the fact that Pott definitely states, " Other people have cancer of the same parts ". Earle relates the case of a gardener who was admitted to St. Bartholomew's Hospital with " a large cancerous sore, which reached from the bend of the wrist to the knuckles, occupying almost the whole of the back of the left hand ". The patient had been engaged in scattering soot in the garden, and eventually the hand was amputated. The specimen is still preserved in the museum at St. Bartholomew's Hospital (Specimen No. Z268).

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by volume. Further increase in the fuel-air ratio to 0·0716 caused the exhaust to smoke and the carbon monoxide proportion to rise to 1·2%, whereas the carbon dioxide percentage fell slightly. Under these conditions the engine developed 16% more power than it did at the makers' maximum suggested fuel-air ratio.

"The Occupational Hazards of Agriculture" were the subject of a paper by E. F. Edson, of Fisons Pest Control Ltd. Information was presented on the size of the agricultural industry in England and Wales. Typical farm operations were illustrated by slides, emphasizing factors such as isolation, lack of direct supervision, and other risky circumstances inevitable in farm work. An analysis of the annual 130 fatal accidents and 20,000 accidents causing loss of time in farming was given and reference made to the basic healthiness of the farming community.

After lunch the chair was taken by Dr. O. G. Edholm.

J. E. Cotes, F. Meade and M. E. Wise, of the M.R.C. Pneumoconiosis Research Unit, described an analysis of "Treadmill Walking as a Form of Exercise". V. Timbrell, also of the Pneumoconiosis Research Unit, then presented a communication on "Some Physical Aspects of Ballistocardiography". Some aspects of the theory of the separated-table ballistocardiograph were considered. The table moves under the action of the pulsating force produced by the motion of the blood in the subject lying on it. In order that the ballistocardiogram, which is the record of the displacement of the table with time, may represent the impressed force, the ballistocardiograph must possess certain characteristics. The characteristics are those of a simple vibrating system with a high natural frequency and a damping factor of about 0·5. The practical ballistocardiograph must be regarded as a complex system; for example, the frame may not be sufficiently rigid and may vibrate. The characteristics must be determined experimentally and the system modified until the desirable characteristics are obtained. To enable cardiac output to be estimated from the ballistocardiogram, the sensitivity of the system is set to some standard value. For this purpose a mechanical oscillation is employed which applies a sinusoidal force of constant amplitude at a frequency of 2½ cycles per second to the total loaded with dead weight. Such oscillation serves as a subject of constant cardiac output.

"Age and Learning; Theory and Needed Research" was the title of a paper by A. T. Welford, of the Nuffield Foundation Research Unit into Problems of Ageing, Cambridge.

After an interval for tea Dr. T. Bedford took the chair. K. J. Collins, of the M.R.C. Climate and Working Efficiency Research Unit, discussed the problem of "The Corrosion of Metal by Palmar Sweat". A large number of manufacturing, assembling, and packing operations involve handling, with the concomitant risk of contamination by palmar sweat. Sweat residues are particularly troublesome in the case of highly finished metal surfaces and subsequent corrosion is rapid. Some individuals may handle degreased metal parts without causing tarnishing, while others cause considerable corrosion. There is variation in this effect, both from person to person and with the same person at different times. The results of preliminary investigations suggested that the quantity of sweat produced is an important factor in determining the extent of corrosion. However, differences in the amount of corrosion were not always related to individual rates of sweating, but might also be attributed to variations in the composition of palmar sweat. An estimate of the corrosive effect of a number of sweat constituents was obtained by placing drops of solutions containing known physiological amounts of solute on polished steel. Severe rusting was caused by concentrations of inorganic salts similar to those reported by Lobitz and Osterberg (1947) in palmar sweat. Other sweat components, particularly nitrogenous compounds such as urea, were also active in solution. Experiments with artificial sweat solutions indicated that corrosion was enhanced at acid *p*H as compared with neutral or alkaline solution. Special sweat-removing measures are not commonly used, although sweat residues can be removed by aqueous alkaline or emulsion cleaners. Contamination may be prevented by the use of rubber gloves or forceps, but such expedients are frequently unsuitable. Certain barrier creams have been tested and appear to be effective, to some extent, in reducing corrosion although, apparently, only in those cases where palmar sweating is slight.

An abstract of a paper entitled "The Absorption and Elimination of Isotopes of Inert Gases, with Special Reference to Krypton", was read on behalf of J. E. S. Bradley. Classical methods for the estimation of body fat in man are indirect, laborious, and of low precision, as has been shown by the work of Keyes. Moreover, they are difficult to validate precisely in those conditions where the results are of most interest. In consequence, several investigators have sought to use substances having a high solubility in fat, in order to obtain a more direct estimate. The work of Loesser with cyclopropane is particularly important in this field. Previous methods were reviewed, and a brief description of the technique developed for use with radioactive isotopes of inert gases, together with some preliminary results, were given. For purely technical reasons krypton had been mainly employed, although xenon would have been preferable from some points of view. Some more general implications arising from the operations of the atomic energy industry were drawn.

The meeting concluded with a general discussion on the problem of "Heat Stress in Industry". The discussion was opened by J. Weiner, who described the results of some observations made by himself and his colleague, A. R. Lind, of the M.R.C. Climate and Working Efficiency Research Unit, followed by a full discussion of this problem of general interest.

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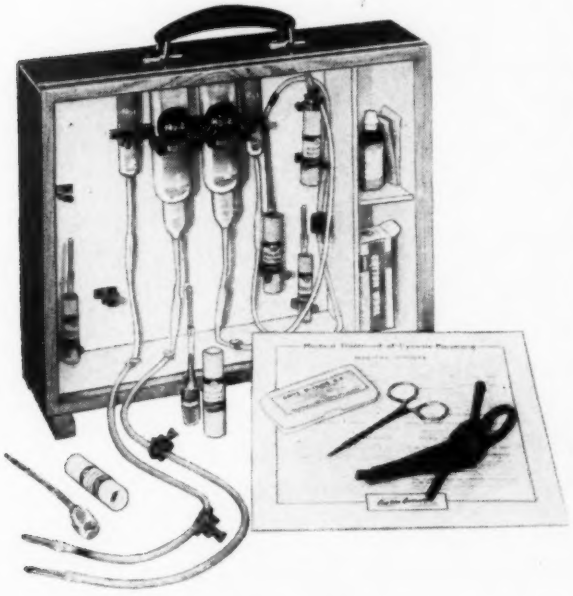

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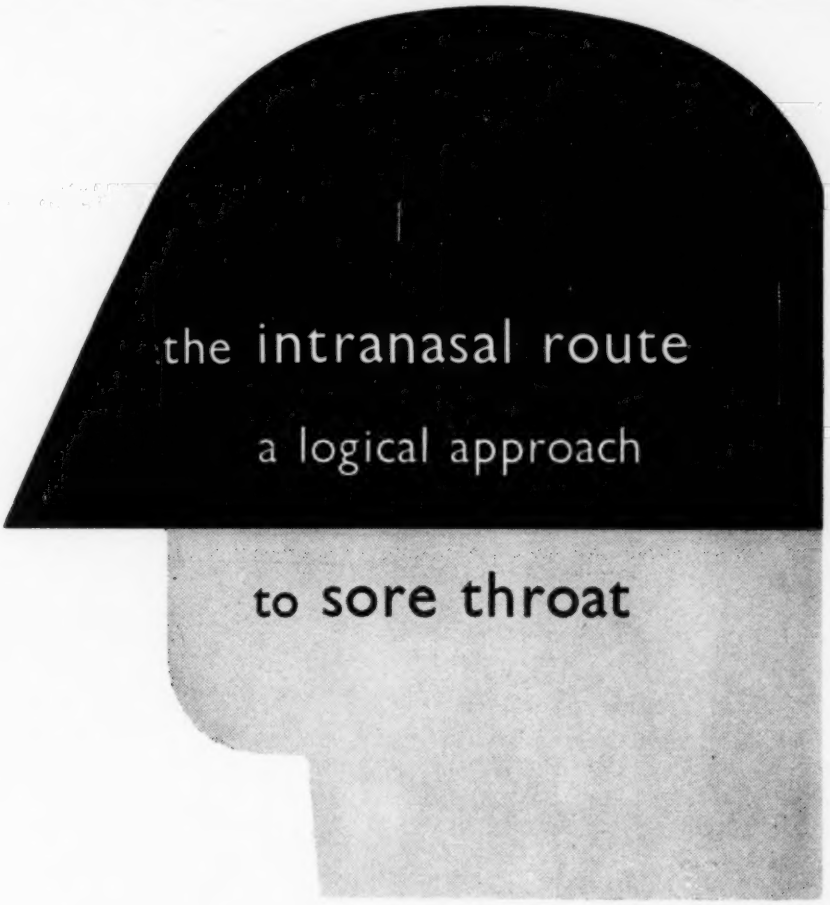

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